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THE
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PRIMARY TUMORS OF THE VERMIFORM APPENDIX: A CLINICOPATHOLOGIC APPROACH

THOMAS W. BOTSFORD, M.D., WM. E. JAQUES, M.D.
ROY CLAUS, M.D.

Boston, Mass.

The management of primary tumors of the vermiform appendix seldom presents a problem because the condition is so infrequently encountered. The problem does occasionally arise however, and for two reasons can be a difficult one to resolve. First, the decision whether the appendiceal tumor is *malignant* or *benign* based upon the usual histologic study is complicated by the fact that argentaffinomas may invade the submucosa, muscularis, serosa, and lymphatics.[11] Furthermore, even in argentaffinomas growing in metastatic foci the tumor cells appear benign by the ordinary cytologic criteria. Second, the number of bona fide primary carcinomas of the vermiform appendix recorded to date are few indeed.[3] Therefore knowledge of the biologic behavior of these tumors is scanty and it follows that the therapeutic experience with such tumors is not large. Because of these considerations, which arose from a recent experience of ours, we wish to present our observations and conclusions in regard to the management of primary tumors of the vermiform appendix. This clinicopathologic study is based upon all the instances of primary appendiceal tumors seen at the Peter Bent Brigham Hospital, Boston, between 1913 and 1954.

This series consists of 34 primary tumors of the vermiform appendix which were selected after a critical review of the tissue sections and clinical protocols of all cases with a previous diagnosis of appendiceal tumor. Many appendiceal lesions were excluded from this series because the appendix was not the primary site of the tumor. Masson[9] in 1928, emphasized the distinction between argentaffinomas (carcinoids) and carcinoma, and it thus became necessary to reclassify many of our carcinomas as argentaffinomas in this study. The majority of the tumors were argentaffinomas as would be expected[13] and they were much more

From the Departments of Surgery and Pathology of the Peter Bent Brigham Hospital and Harvard Medical School.

TABLE 1

Primary lesions of the vermiform appendix

Type	Number	Female	Male
Argentaffinoma (carcinoid)	27	23	4
Adenocarcinoma	3	2	1
Mucocele	3	2	1
Fibroma	1	0	1
Total	34	27	7

common in the female (table 1). This seemingly high incidence of argentaffinomas in the female is undoubtedly due to the universal practice of incidental appendectomy in gynecologic operations. The argentaffinomas were evenly distributed through all age groups, 18 to 60 years, but the 3 adenocarcinomas in this series occurred in patients who were over 40 years old.

OBSERVATIONS ON THE HISTOLOGY OF APPENDICEAL TUMORS

Argentaffinomas

The microscopic characteristics of the argentaffinomas did not differ from that described by previous observers,[11] so only a brief summary of the more important features will be mentioned here. The tumor characteristically grew in cords,

FIG. 1. Argentaffinoma showing bundles, cords and abortive acinar formation. The groups of tumor cells are separated by relatively acellular connective tissue. (Hematoxylin-Eosin × 212).

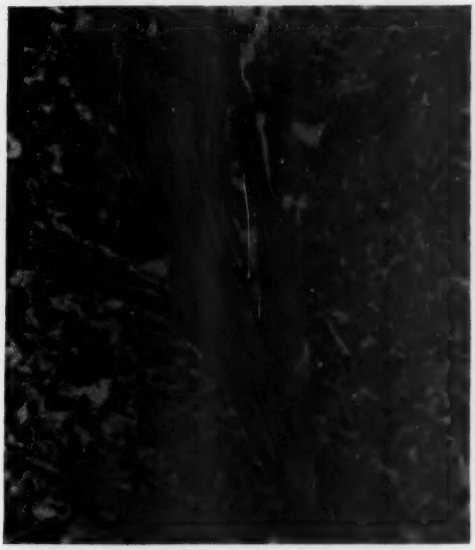

FIG. 2. Argentaffinoma invading the muscularis externa. The groups of tumor cells can be seen interspersed between the bundles of smooth muscle fibers. (Hematoxylin-Eosin X 212).

bundles, finger-like projections and isolated groups of cells (fig. 1). Small acini and abortive acini occasionally were seen, but were easily differentiated from the primary adenocarcinomas of the appendix by their general growth pattern. The tumor cells frequently were distorted as they invaded the muscularis externa (fig. 2). In this latter location, the tumor cells did not invade the muscle fibers but appeared to penetrate between muscle fibers. They generally were polygonal with indistinct cell borders, and their nuclei generally were characterized by a powdery chromatic network. Fine vacuolation of the cytoplasm was frequent, but could be easily separated from the large mucous vacuoles of the adenocarcinomas. In a few cases, a fine yellowish-brown pigmentation of the cytoplasm was noted. In the sections stained with silver, we found this to be present (fig. 3). There generally was a marked desmoplastic reaction to the tumor. By means of special stains, it was found that fibrous connective tissue, elastic fibers, reticulum fibers, smooth muscle fibers and occasionally nerve fibers contributed to this process. A rare mitosis was encountered (fig. 4). The cells generally did not vary a great deal in size, shape or tinctorial qualities. Rarely, hyperchromatism was observed. No definite lymphatic or vascular invasion was encountered.

The histologic grading of argentaffinomas is a moot question. They originally were considered to represent a benign tumor.[9] In more recent years a number of observers have stressed the malignant potentialities of these tumors, especially the nonappendiceal variety.[11, 2, 1, 12, 4, 10] We have classified the argentaffinomas in relation to their location in the coats of the appendiceal wall (table 2). It is of

FIG. 3. Silver particles can be seen in the cytoplasms of the cells constituting an argentaffinoma. Argyrophilic fibers are noted surrounding the group of tumor cells. (Silver stain after the method of Rogers × 1030).

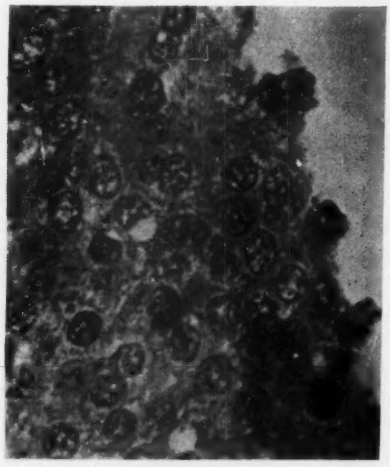

FIG. 4. Close up of a group of tumor cells in an argentaffinoma. A mitosis can be seen. The tumor cells are uniform in size, shape and tinctorial qualities. (Hematoxylin-Eosin × 600).

interest to note that 17 argentaffinomas of our series would be categorized as invasive and that the serosa was involved in 10 instances (groups 3 and 4, table 2), and in 1 instance the tumor had also invaded the mesoappendiceal fat.

TABLE 2

Local anatomic limits of appendiceal argentaffinomas

Location and Extent	Number of Cases
1. Mucosa only	1
2. Mucosa and submucosa	9
3. Mucosa, submucosa and muscularis	7
4. Mucosa, submucosa, muscularis and serosa	10
Total	27

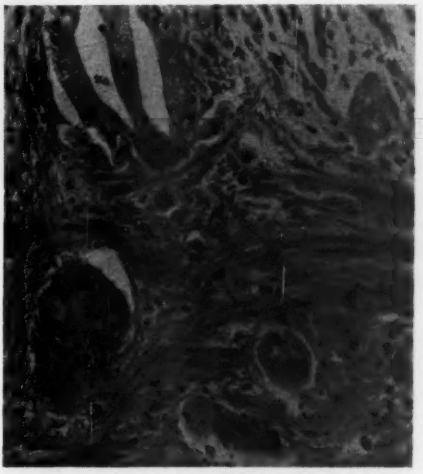

FIG. 5. Adenocarcinoma invading the muscularis external of the appendix. A definite acinar configuration is apparent. A large mucus vacuole is also noted. (Hematoxylin-Eosin × 212).

Carcinoma

We found it comparatively simple to separate the carcinomas from argentaffinomas as 2 of them closely resembled their counterparts arising in the colon. Acinous formation was the rule with not infrequent papillary projections. The acini were larger, more numerous and better developed than in the argentaffinomas. In each case, it was possible to identify large mucous vacuoles in the cytoplasm of the tumor cells (fig. 5). Although the argentaffinomas displayed finely vacuolated cytoplasms, no large mucous vacuoles were encountered. Much more pleomorphism was noted in the cells composing this neoplasm and mitoses were more frequent. In every case, the tumor extended from the mucosa to serosa. One case represented, in our opinion, an adenocarcinoma arising in a mucocele. This lesion caused attenuation of the appendiceal wall. It was an infiltrating tumor lined by tall secretory epithelium and producing abundant mucus replacing the remainder of the appendix (fig. 6).

FIG. 6. Details of an adenocarcinoma arising in a mucocele of the appendix. A large acinus displaying papillary projections and lined by mucus secreting epithelium is noted. (Hematoxylin-Eosin × 212).

FIG. 7. Gross appearance of a mucocele of the appendix. There is a bulbous dilatation of the distal portion of the appendix. This case was complicated by the finding of endometrial implants over the serosal surface of the mucocele and in the mesoappendiceal fat.

Mucocele

The mucoceles presented an essentially similar picture in all instances. They were located in the tip of the appendix and caused attenuation of the appendiceal wall in 2 instances. In each case, the mucocele was partially lined by hyperplastic secretory epithelium. The epithelium was regular in appearance and no invasiveness was encountered. There was an accompanying chronic inflammatory

process in the wall in each case. In 1 instance fibrinopurulent exudate was noted over the serosal surface. Copious quantities of mucus were present in many areas and the lining was devoid of any epithelium. Waugh and Findley[14] described a case in which apparent cancerous transformation occurred in a mucocele. Such was the case in 1 of our adenocarcinomas mentioned above.

Fibroma

The single fibroma presented as a nodule measuring 1 cm. in diameter and located 2 cm. from the appendiceal base, and was an incidental finding. It was firm in texture and white in color. On microscopic section, it was eccentrically located in the submucosa and produced pressure atrophy of the surrounding structures. It was dense, relatively acellular and no neurogenic elements were identified.

CLINICAL OBSERVATIONS ON APPENDICEAL TUMORS

Argentaffinomas

Six of our 27 patients with appendiceal argentaffinomas had appendectomies because of acute appendicitis. Their symptoms were in no way different from those of other patients with appendicitis. In 2 of these patients with acute appendicitis the tumor was situated in the extreme proximal portion of the appendix and was transected when the appendix was removed. Histologic examination showed the tumor to have invaded all coats of the appendix. Resection of the cecum, part of the ascending colon, and a portion of the terminal ileum with ileocolostomy was done in these 2 patients. No residual tumor could be found in 1 specimen, while in the other an argentaffinoma was found in the appendiceal stump and was invading the cecum. No lymph node metastases were found. The remaining argentaffinomas were found in appendices incidentally removed in 19 instances and at postmortem in 2 patients. The tumor was well away from the cut proximal portion of the appendix in every instance except the 2 cases mentioned above. Follow-up studies on all patients with appendiceal argentaffinoma, from 1 to 15 years postoperatively have shown no evidence of further disease related to the primary tumor of the appendix.

Carcinoma

The 3 instances of carcinoma of the appendix will be briefly described in individual cases.

CASE REPORTS

Case 1. M. W., a 55 year old white woman, entered the hospital because of symptoms of appendicitis of 24 hours duration. She had been in previous good health. A diagnosis of acute appendicitis was made and a laparotomy was done. A perforated inflamed appendix was removed and a large abscess was drained. The patient was quite ill postoperatively, but finally improved. The pathologist reported that the appendix contained adenocarcinoma. She was sent home to convalesce and advised to return to the hospital for a right colectomy. She did not return to the hospital and died at home six months after the appendectomy.

Comment: This carcinoma of the appendix fulfilled all the histologic criteria for malignancy. The extra-appendiceal extent of the tumor could not be determined at operation, but the operator was certain the cecum was not involved. No postmortem examination was made.

Case 2. L. L., a 41 year old white man, entered the hospital because of symptoms referable to acute appendicitis of 48 hours duration. An acutely inflamed perforated appendix was removed and the abdomen was drained. After a stormy postoperative course the patient recovered and was discharged home. Because of the histologic diagnosis of adenocarcinoma of the appendix, he was advised to return to the hospital for a right colectomy. He never returned to the hospital and all immediate and late follow-up queries were unanswered.

Comment: This patient had a diagnosis of primary carcinoma of the appendix as well as acute appendicitis. The surgeon was certain that the cecum was not involved by the tumor.

Case 3. J. W., a 52 year old white woman, entered the hospital because of abdominal pain of several months duration. Exploratory laparotomy was done. The following is quoted from the surgeon's operative note. "The appendix presented a remarkable appearance. Its outer third was apparently the seat of a cystic process which had burst, and there was adherent about the opening a mass of the same gelatinous material present in the omentum. The proximal portion of the appendix contained a cyst-like mass which actually projected into the cecum. Apparently this was the original site of the process." This patient had no further surgery for the appendiceal lesion except appendectomy. One year later she had a cholecystectomy and at that time tumor implants were noted over the serosal surfaces of the liver and intestines. She died two and one-half years after the appendectomy. No postmortem examination was made.

Comment: The patient had a primary appendiceal carcinoma which probably arose in a pre-existing mucocele.

Mucocele

Three of our patients had benign mucoceles; 2 mucoceles occurred in patients who had complained of vague chronic lower abdominal pain. The disease was not diagnosed preoperatively but was found at exploratory laparotomy. The other mucocele was found in a patient who had endometriosis and who was admitted to the hospital because of acute abdominal pain. A diagnosis of acute appendicitis was made and at operation a mucocele covered with endometrial implants (fig. 7) was found and removed by appendectomy.

DISCUSSION

Perusal of published reports concerning primary tumors of the appendix show that considerable confusion exists about the subject. The classification of argentaffinomas, incidence of carcinoma, and treatment of the lesions are the major points of differences of opinion. The terms *carcinoid carcinoma*,[5] *adenocarcinoma, Grade I (carcinoid)*[7] and *argentaffinoma* are used interchangeably, but we are of the opinion that argentaffinoma is the preferable term to use. In 1942, Young and Wyman[15] reported 1 primary appendiceal cancer and found only 4 similar cases in the literature. In 1949, Lesnick and Miller[8] in a critical review

reported 17, and in 1953, Clarke and Simonds[3] collected only 31 cases. Hilsabeck[6] recently reported 12 instances of primary carcinoma of the appendix, 3 of which arose from adenomatous polyps of the appendix.

It can be assumed that authentic primary carcinoma of the appendix is a rare disease. Carcinoma of the cecum which has involved the appendix should not be categorized as a primary appendiceal tumor. Because of its infrequent occurrence, an extensive experience has not accumulated on the management of primary cancer of the appendix. Opinions on the treatment of primary cancer of the appendix vary from those who advocate appendectomy only to those who recommend right colectomy.

After a study of the literature and from our own experience, we are of the opinion that the problem can be solved by basing treatment on the histologic findings and proceeding by the following principles. It can be assumed that the diagnosis of primary appendiceal tumor is rarely if ever made prior to operation. The surgeon removes the appendix because of acute appendicitis or incidentally at laparotomy for other indication. With the exception of mucocele of the appendix the surgeon may not find gross evidence of appendiceal tumor at the time of appendectomy. It is paramount that all appendixes removed for any cause be examined grossly and microscopically, but frozen section examination is not indicated unless there is tumor grossly present. Because argentaffin tumors may involve only a portion of the circumference it is advisable to open the appendix along its long axis to permit complete study of the entire mucosal surface. Small argentaffin tumors may be overlooked if the appendix is transected. This is borne out by the data in table 2 where it is noted that in only 10 of 27 argentaffin tumors was the serosa involved. It is occasionally difficult to distinguish between argentaffinoma and carcinoma and in addition special staining of the tissue often is required. The definitive diagnosis of the appendiceal tumor then comes from the pathologist several days later. It is at this point that a decision must be made as to further management.

If a histologic diagnosis of carcinoma of the appendix is unequivocally made, the patient should be advised to have a right colectomy. Despite isolated reports of *cures* of proved primary appendiceal cancer by appendectomy alone, there are many who were not cured by that procedure.[8] Furthermore, in the confusion that has existed in the histologic differentiation between argentaffinomas and carcinoma, *cures* by appendectomy alone should be viewed with a critical eye. Finally, observation of a malignant tumor of the gastrointestinal tract is a dangerous procedure at best. Indications of progression of such a tumor usually mean the patient is incurable.

Appendectomy generally is sufficient treatment for appendiceal argentaffinomas. These lesions most commonly are situated in the distal part (tip) of the organ[4] which may explain why they rarely metastasize. If, however, the lesion is at the base (proximal portion) of the appendix and tumor is transected by the appendectomy and the lesion is invasive (table 2), thorough consideration should be given to further surgical excision.

Mucoceles of the appendix should be treated on the basis of their gross and

microscopic appearances. Thus an unruptured *cyst* of the appendix without microscopic evidence of carcinoma would be treated by appendectomy. If signs of invasiveness are present more radical surgery is indicated. According to others,[14] if the mucocele has become invasive it does so by involvement of nearby organs and the production of pseudomyxoma peritonei.

SUMMARY

Thirty-four instances of primary noninflammatory lesions of the vermiform appendix are reviewed. The histologic features of 27 argentaffinomas, 3 carcinomas, 3 mucoceles and 1 fibroma are discussed.

Three instances of authentic primary cancer of the vermiform appendix are reported.

The clinical manifestations of appendiceal tumors are discussed, and recommendations for the management of the different types of primary tumors of the vermiform appendix are made.

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INDICATIONS FOR SURGICAL TREATMENT OF DUODENAL DIVERTICULA

ROBERT WOODRUFF, M.D.

Denver, Colo.

Duodenal diverticula are relatively common anatomic variations in the human intestinal tract. Case[1] apparently was the first to make a roentgenologic diagnosis of this entity. Since his report in 1916 there have been many references to this interesting lesion. The reported incidence varies between about 1 and as high as 20 per cent. The diverticula are found less commonly by roentgenogram than by autopsy studies. Cattell and Mudge[2] reported an incidence of 1.9 per cent of duodenal diverticula in a series of 2000 consecutive gastrointestinal examinations studied roentgenologically at the Lahey Clinic. Horton and Mueller[5] found duodenal diverticula in 5 per cent of 216 consecutive autopsies at the Mayo Clinic.

About 75 per cent of true or primary diverticula are found in the second part of the duodenum. The rest are scattered throughout the other parts with the exception of the first part where they are rare. Diverticula in the first part are most often secondary to inflammatory changes associated with duodenal ulcer. Characteristically, the classical lesion arises from the medial part of the second portion closely associated with the ampulla of Vater. Most often it comes off posteriorly and extends into or behind the head of the pancreas. Less often the pouching comes off anteriorly. It is this close association with the important biliary and pancreatic structures that makes the removal of this lesion so hazardous.

The theories of origin are usually based on increased intraluminal pressure with herniation through the wall at congenital weak points which are more commonly found on the medial side of the second portion of the duodenum at the points where the wall is traversed by blood vessels, nerves, and ducts. The true diverticula are usually deficient of muscular coat and are characteristically made up of mucosa and submucosa covered over by the serosal layer.

It is generally agreed that by far the majority of these lesions are of only academic interest and that it is only the occasional duodenal diverticulum that is of sufficient clinical significance to warrant its surgical removal. When one is cognizant of the real hazards in the technical removal of these lesions, as evidenced by two deaths from enzyme leaks and pancreatitis in the series from the Lahey Clinic,[2] he is reluctant to undertake the procedure without good evidence that the lesion is responsible for the patient's symptoms and trouble. On the other hand it is most important to recognize the significance of the lesion and not ignore it when it is really at fault. Patterson and Bromberg[7] report a case in which a gallbladder with stones was removed, only later to find that the

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real cause of the patient's trouble was a duodenal diverticulum which was originally thought to be asymptomatic. It would be equally easy to attribute gastrointestinal symptoms to a simple diverticulum which in reality were caused by a much more serious condition such as an early malignancy.

It is apparent that the diagnosis of these lesions is almost entirely in the hands of the roentgenologist. Occasionally a duodenal diverticulum may be found on exploration of the abdomen, but that would certainly be the exception in as much as most of these lesions are not exposed until the duodenum has been reflected. The problem that falls to the clinician and surgeon is the decision as to which of these lesions is clinically significant and causing sufficient trouble to justify consideration of surgical extirpation.

Various classifications of duodenal diverticula have been proposed from time to time. These generally have been attempts to segregate the asymptomatic group from those that are of true clinical significance. In many instances these classifications seemed to have been based on the clinical manifestations which were most prominent in the author's individual cases. It would seem more rational to approach the problem by classifying according to actual pathologic change in the lesion itself and by the distortion of function brought about by actual stasis in or distension of the lesion itself. Accordingly the following classification is proposed:

I. Diverticula with primary pathologic changes.
   A. Inflammatory.
      1. Diverticulitis with or without perforation and peritonitis.
      2. Ulceration and erosion with pain and/or hemorrhage.
   B. Neoplastic.
II. Diverticula with stasis or distension.
   A. Stasis causing gastroduodenal dysfunction.
   B. Distended diverticula interfering with biliary, pancreatic and duodenal function.

From a review of the literature and my own experience, I am impressed with the fact that the majority of the symptomatic diverticula fall into the group with stasis or distension, and it is here that we encounter the real problem in selecting cases in which surgery is indicated. Diverticula with primary pathologic changes are encountered less commonly, and generally it would be expected that the diagnosis would be apparent. The exception may be in a case in which a diverticulum was being considered as a source of gastrointestinal bleeding. A case in point will be presented.

My experience with the surgical treatment of duodenal diverticula has been limited, but it is believed that a presentation and evaluation of personal cases is of value in this relatively uncommonly encountered clinical entity.

CASE REPORTS

Case 1. A 47 year old white man was first seen in February 1944. His only complaint was weakness and other signs of anemia. This was the result of repeated episodes of gastrointestinal hemorrhage manifested by tarry stools over the previous several months. He had no other significant gastrointestinal symptoms and had always been in good health prior to

the present trouble. Roentgenographic study of the entire intestinal tract was within normal limits except for a diverticulum protruding from the medial side of the second portion of the duodenum. Sigmoidoscopic visualization did not reveal anything of significance. Because the patient continued to bleed under observation, and because there was nothing else to explain the clinical findings, the diverticulum was incriminated and on March 3 the abdomen was explored. No pathologic change was noted in any part of the abdomen except on the second portion of the duodenum. Superficial inspection and palpation revealed nothing and it was not until the duodenum had been reflected medially, after incising the lateral peritoneum, that the lesion was brought into view. The diverticulum measured about 2 cm. across its base and about 3 cm. in length. The proximal part of the base was about 1 inch below the ampulla of Vater. The pouch appeared to protrude through a defect in the muscular coat and grossly it was devoid of that layer. It was mobilized quite easily under direct vision and clamped flush with the duodenal wall. The clamp was oversewn with a row of continuous chromic catgut sutures and the closure was completed with a row of interrupted mattress sutures of cotton placed in the seromuscular layer. The patient made an uneventful recovery and one year later he was well and had had no further evidence of gastrointestinal bleeding. Pathologic examination did not reveal any open ulceration in the mucosa of this diverticulum. However, it was believed justifiable to assume that the diverticulum was the cause of the bleeding in as much as there was nothing else to explain it and in view of the fact that the patient remained well following its removal.

FIG. 1. The roentgenogram demonstrates a long diverticulum arising from the medial side of the second portion of the duodenum.

Comment. Marshall[6] states that duodenal diverticula are a cause of gastroduodenal bleeding and believes it is due to erosion of vessels following inflammation. Frank[4] has reported a well substantiated case of bleeding due to a duodenal diverticulum in which the specimen did not show a definite bleeding point.

The diverticulum in this case arose from the posterior and medial portion of the second portion of the duodenum and, as mentioned, in that position was not readily accessible to identification until the duodenum had been reflected and mobilized. This shows the futility of expecting to find a lesion like this by routine abdominal exploration and reiterates the necessity of careful roentgenologic examination if the lesion is to be readily found. In this case, and in the others in my experience the lesion was readily dissected under direct view, and it was not considered necessary to open the sac, as suggested by Pearse[8], during its removal or to resort to inversion recommended by Ferguson[3] to prevent injury to neighboring structures. However, one would do well to be familiar with these technics should a lesion be encountered in which the direct removal would be fraught with danger because of inflammation and distortion of structures.

Case 2. A 33 year old white man was seen in December 1944 complaining of acute epigastric pain with localization to the right of the midline. He stated that he had had repeated similar episodes of pain in the past, and that several weeks earlier he had had an appendectomy during one of these attacks. He stated that following the operation the pain seemed to be relieved for a while, but again it began to flare up as before. There was moderate tenderness which was localized just to the right of the umbilicus. Further examination, which

FIG. 2. Same patient as figure 1, 24 hours after the ingestion of barium. The diverticulum has trapped a considerable quantity of the contrast media.

included roentgenologic study of the stomach and duodenum, revealed a diverticulum of the second portion of the duodenum (fig. 1). It is important to note that the diverticulum retained barium for 24 hours (fig. 2). Operation was done on December 22, and, as in the first case, superficial inspection of the duodenum was not conclusive. The peritoneum along the lateral side of the second portion of the duodenum was incised and as the organ was reflected medially the diverticulum was encountered coursing distalward from its origin on the medial and posterior wall of the bowel under the duodenum toward the right lower quadrant. It was mobilized and removed in the same manner as in the first case. The diverticulum was about 2 cm. in width and measured about 6 cm. in length. It contained barium and a moderate quantity of partial digested food. The patient made an uneventful recovery and when last heard from had had no further abdominal pain. There were only minimal inflammatory changes in the diverticulum walls.

Comment: This case represents a lesion with the clinical symptoms due to stasis and distension. The symptoms were undoubtedly due to accumulation of gastroduodenal contents in the out-pouching. The intermittant nature of the pain in this case deserves comment. It was probably due to the periodic filling of the lesion which has been commented on by others. When present it is helpful in determining whether or not the patient's symptoms were caused by the

FIG. 3. The diverticulum is noted originating from the terminal anomalous duodenum as described in the text (case 3).

duodenal diverticulum. This case further illustrates the danger of attributing symptoms of diverticula to some other pathology—in this case appendicitis.

Case 3. The third case was a 33 year old white man, a truck driver, who was first seen in consultation on Nov. 24, 1952. For the previous seven to eight years he had had attacks of epigastric pain which had gradually become more persistent and severe. Again the intermittent nature of the symptoms is of importance. There was occasionally temporary incomplete relief after eating, but generally the intake of food aggravated the pain. He was particularly prone to have the symptoms while engaged in his occupation of driving a truck transport. He stated that at times the jarring of the vehicle aggravated the pain to such an extent that he had to stop by the roadside for relief. He doubted that he would be able to continue in his occupation unless relief was obtained. Gastroduodenal roentgenograms showed the diverticulum illustrated in figure 3. It was the opinion of the radiologist that the lesion arose in the second part of the duodenum. Figure 4 shows retention of barium after 24 hours.

On November 28, the abdomen was explored. An anomalous arrangement of the distal portion of the duodenum was noted. Instead of coursing medially, the third portion of the duodenum started posteriorly, then went superiorly under the second part and at the upper portion of that part coursed medially under the duodenum and pancreas across the spine to take up its normal position. The second portion was reflected medially and the usual site of the lesion carefully explored without success. The radiologic pictures were re-evaluated and then it was decided that the diverticulum was arising from the distal duodenum medial to

FIG. 4. Same patient as figure 3. Roentgenologic evidence of retention in the diverticulum after 24 hours is demonstrated.

the descending duodenum in such a manner that it was confusing in the picture as to just where the lesion arose. That portion of the duodenum was then mobilized and the diverticulum was removed as in the previous cases. It measured about 2½ by 4 cm. and the neck was 12 mm. in width. There was nothing remarkable noted on pathologic examination. The patient has subsequently returned to his old work and has remained free of epigastric pain.

Comment: Again this case is probably an example of a diverticulum in which stasis was the cause of the clinical picture. The condition was aggravated considerably by the patient's occupation as a truck driver. The trapping of the barium in the diverticulum was of considerable help in deciding on surgical treatment and it is believed that demonstration of stasis in the diverticulum should be a requisite for diagnosis of duodenal diverticulum with symptoms due to stasis or distension. It has been repeatedly commented on in the literature that duodenal diverticula are more often found in the older age groups. It is interesting that these 3 cases represent relatively young individuals.

SUMMARY AND CONCLUSIONS

A short review of duodenal diverticula has been presented. It has been noted that this is a rather common entity demonstrated by both radiologic and autopsy study.

I am in complete agreement with most other authors that this lesion is only occasionally of clinical significance. However, I have endeavored to stress the importance of implicating this structure and treating it properly when it is responsible for clinical disturbances.

The technical hazards of dealing with the lesion precludes unnecessary meddlesome surgery, but, on the other hand, the danger of serious clinical disturbances demand treatment when indicated.

The danger of implicating other structures when the diverticulum is at fault is commented on.

A clinical classification is proposed to aid in the diagnosis. Epigastric pain intermittent in character in my opinion is the most pathognomonic symptom and roentgenographic evidence of barium retention is the most helpful finding in selecting cases for operation.

Three personal cases are briefly presented to illustrate important points in diagnosis and management.

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SURGICAL DISEASES OF THE PULMONARY ARTERY: CENTRAL ANEURYSMS, ARTERIOVENOUS FISTULAS, AND PERIPHERAL ANEURYSMS

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In a series of 109,571 autopsy examinations, aneurysm of the pulmonary artery was encountered and described only eight times. This represents a ratio of 1 case in 13,696 autopsies.[12] Different authors who have reported large series of arterial aneurysms, even when the lesions have been restricted to those involving the intrathoracic vessels, only rarely report an aneurysm of the pulmonary artery. At the Mayo Clinic from 1925 to 1935, of 596 arterial aneurysms, 339 of which were intrathoracic, no aneurysms of the pulmonary artery were found.[26] Current interest is focused not only on the vascular lesions themselves, but also on lesions which easily may be confused with them.

Aneurysm of the pulmonary artery is a permanent, more or less circumscribed, dilatation of any portion of the pulmonary artery associated with organic degeneration of the vessel wall. Dilatation of the pulmonary artery has been a surprisingly frequent finding in many diverse conditions, but destruction of one or more layers of the vessel wall must be demonstrated before it can be described accurately as an *aneurysm*. Simple dilatation of the pulmonary artery occurs in mitral stenosis, pulmonary arteriolar sclerosis, and some congenital anomalies such as (a) interauricular septal defect; (b) patent ductus arteriosus; (c) interauricular septal defects with mitral stenosis; (d) Eisenmenger's complex; (e) Laubry's syndrome (unequal division of the truncus arteriosus with resulting large pulmonary artery and small aorta); (f) Lutembacher's syndrome (largest pulmonary arteries occurring in congenital lesions); (g) isolated pulmonic stenosis; and (h) aberrant return of pulmonary veins.

Aneurysms of the pulmonary artery may be divided into those involving the trunk and the main stems of the pulmonary artery, pulmonary arteriovenous fistulas, and aneurysms involving the peripheral branches of the pulmonary artery deep in the pulmonary parenchyma. In these cases the trunk and stem branches are almost exclusively involved, while only a small per cent (circa 15 per cent) involve the peripheral branches.

In a series of 108 patients with central aneurysms of the pulmonary artery, a sex ratio of 1 to 1 was observed.[7] This contrasts markedly with the ratio of 5.6 males to 1.0 females observed in cases of aortic aneurysms. The average age for the entire series was 37.7 years for the females, and for the males 37.2 years. Thirty per cent of the patients were less than 30 years of age, compared with 15 per cent of patients in this category in the case of aortic aneurysm. The preponderance of cases involving the main trunk is attested to in all series. Such

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involvement is stated to occur in about 85 per cent of cases. In 36 cases of aneurysm of the pulmonary artery verified by necropsy, Deterling[12] found that the trunk was affected, with or without involvement of the major branches, in 89 per cent, while the right branch was involved in 8 per cent and the left branch in 3 per cent. In these 36 cases, the aneurysm was fusiform in 24, and saccular in 12. On the other hand, Boyd and McGavack[7] reported a ratio of fusiform to saccular aneurysm of 3 to 4. Syphilis was considered to be a positive etiologic factor in 31.7 per cent of Boyd and McGavack's series, while Deterling considered that syphilis played a probable role in 39 per cent. This is in contrast to the stand taken in 1917 by Warthin,[34] who attributed all cases of this lesion to syphilitic involvement. In both series a patent ductus arteriosus was by far the commonest cardiovascular anomaly associated with pulmonary aneurysm. This occurred in 21 per cent in Deterling's series and 23 per cent in Boyd and McGavack's series. The next commonest anomaly was an interventricular septal defect which was found in 4 of 36 cases in Deterling's series. One case each of aortic hypoplasia, bicuspid pulmonary valves, and doubly branched subclavian artery were found in this same series. Mycotic degeneration of the wall of the pulmonary artery is usually associated with an infected patent ductus arteriosus or subacute bacterial endocarditis with valvular vegetations. Much more commonly mycotic aneurysms involve the peripheral branches of the pulmonary artery. Pulmonary atherosclerosis secondary to physiologic alterations of the pulmonary circuit has been observed. In some cases there has been marked arteriolar sclerosis throughout the lung with right cardiac dilatation.

Exertional dyspnea is the most common manifestation of aneurysm of the pulmonary artery. Associated with it are cough, occasional hemoptysis, and thoracic pain. Most commonly this pain is precordial with occasional extension to the shoulder and arm. In cases in which the aneurysm has enlarged suddenly or has undergone rupture, severe, sharp pain followed. Cyanosis is usually due to some congenital cardiac lesion which alone is the cause. Unlike pulmonary arteriovenous fistula, aneurysm infrequently causes clubbing of the fingers and toes. In contrast to aortic aneurysm, involvement of the recurrent laryngeal nerve with ensuing hoarseness has never been reported.

Frequently a somewhat harsh systolic murmur heard in the second or third intercostal space at the left border of the sternum has been noted. In a few cases, a separate diastolic murmur also has been present. If the aneurysmal dilatation should be obliterated by an organized thrombus so that eddy currents could not be produced, these auscultatory phenomena would not be audible. Dullness in the second left intercostal space may accompany enlargement of the pulmonary conus of the artery. Right-sided cardiac enlargement is almost constantly present at autopsy, but may elude clinical examination. In 16 cases in which the electrocardiogram was recorded, there was right axis deviation in 12 (75 per cent) (Deterling[12]).

In general, these central pulmonary aneurysms appear on the roentgenogram as discrete, rounded shadows at the pulmonary hilum. By oblique views the shadows can be outlined beneath the aortic arch and anterior to it. Aneurysm

of the aorta, since it is much commoner than pulmonary artery aneurysm, must be carefully excluded. The globular shadow may pulsate if a thrombus has not formed. The left side of the silhouette may move in a to-and-fro manner with the area of the pulmonary conus and the ventricular shadow alternately expanding. In the right anterior oblique view, the aneurysmal shadow may be seen to encroach upon the retrosternal space. It may present a knob-like appearance in the posterior mediastinum below the level of the aortic arch and above the level of the left atrium. In cases in which expansile pulsation is present, this may be transmitted through the lesser pulmonary radicals to result in what has been termed the *hilar dance*.[28] It must be noted, however, that there are cases of aneurysm of the pulmonary artery, confirmed by autopsy, in which no pulsation was seen by radiographic studies.

An aneurysm of the pulmonary artery may resemble a patent ductus arteriosus more than an aortic aneurysm, because it sits further anteriorly, and the aorta may often be well delimited in the oblique views.[33] The size of the pulmonary aneurysm increases with prolonged observation, while the patent ductus arteriosus remains constant, if the patient has reached adult age. Pulmonary aneurysms frequently show calcification of their walls. The pulsation progresses synchronously with that of the aorta. With Valsalva's maneuver, on the other hand, the size of the pulmonary aneurysm increases, while the aortic aneurysms become smaller. In more recent times angiocardiography, pneumomediastinum and roentgen kymography have been added to the available technics and have increased the possibilities of diagnosis of pulmonary aneurysms during life.

While actually radiographically the central pulmonary aneurysm is to be differentiated from the diseases of the mediastinum, the peripheral pulmonary aneurysm belongs in the group of round lung shadows. Originally, the peripheral aneurysms were only recognized as accidental findings at autopsy. Radiologically, they could not be differentiated from the large group of round lung shadows. The major portion of these peripheral aneurysms produce an arteriovenous shunt in the lesser circulation which may have as a consequence a severe disease picture.

An arteriovenous fistula is a communication of an artery with a vein without the interposition of a capillary bed; it arises on the basis of embryologic malformation. This represents initially a simple connection between arteries and veins. By change of the pressure relations in the lesser circulation, indeed, the venous as well as the arterial limb can be widened aneurysmally and reach considerable size to increase the cross-section of the fistula so that there originate sac-like structures with labyrinthine hollow spaces. The origin and increase in size can be observed radiologically. Men and women are equally affected. Arteriovenous fistulas of the lung are often associated with other developmental aneurysms of the vascular apparatus and appear frequently in families. The frequent combination with Osler's disease is striking.[15, 17, 30] A short circuit connection in the lesser circulation must have as a consequence anoxemia of the peripheral blood with dyspnea, cyanosis, clubbing of the fingers, and polycythemia. However, marked anemia due to hemorrhage may act in the opposite sense and obscure the usually very typical signs. After the disappearance of the

anemia, polycythemia may reappear.[2] Half of the cases of arteriovenous fistula show evidence of Osler's disease. This disease picture was described for the first time by Osler in 1901 as telangiectasia herediteria hemorrhagica. The following symptom triad is frequently present: telangiectases of the skin and mucous membranes, hemorrhagic tendency, and inheritance as a dominant gene. Further observations, since the publication of Osler, show that it is a matter not only of telangiectasia of the skin and mucous membranes, but that every organ of the body may be affected by the vascular malformations. Investigations of families with Osler's disease have shown[14] that in one group one finds all typical signs of the disease without pulmonary changes, while individual members of the family show only arteriovenous fistulas of the lung and external telangiectases are lacking. Cases which have been designated as hereditary hemoptysis and some cases of telangiectasis of the bronchial mucosa may in any case be placed in the group of arteriovenous fistulas.

Arteriovenous fistulas occur as round, smoothly delimited, blood-filled nodules in the parenchyma of the lung. They vary in size from 0.5 cm. to 8 cm. in diameter. They have an average diameter of 3 cm. and in about half of the cases they are multiple, distributed over one or both lungs. Up to seven separate lesions have been described in one patient. The lower portions of the lung are most frequently involved, and occurrence at the lung apices has never been described. They lie more frequently at the periphery than at the hilus, in contradistinction to the acquired aneurysms which appear close to the main stems. With increasing size, multilocular sacs are formed. In these saccular, blood-filled, labyrinthine spaces, a dilated pulmonary artery inosculates with a markedly widened pulmonary vein. The walls may be very thin so that the streaming of blood may be observed at operation.[8] Frequently, however, mural thrombi are present which produce thickening of the wall and calcification. Adhesions to the lateral chest wall and to the diaphragm are frequent and make operative intervention difficult.[13] The calibre of the afferent and efferent vessels, which as a rule course spirally toward the hilus, can reach considerable width, up to 2 cm. in diameter. Atypical vessels are not rare, and the artery can arise directly from the aorta while the vein may inosculate into the inferior vena cava.[16, 35] Collateral vessels leading to the thoracic wall may make operation difficult.[9] The surrounding pulmonary tissue is usually normal. With increasing size of the aneurysm, compression atelectasis may appear. Small rents in the wall of the aneurysm may produce hemorrhage into the pulmonary parenchyma.

The communication of a pulmonary artery with a pulmonary vein without the interposition of a capillary bed produces changes in the circulatory dynamics. Blood of the pulmonary artery may stream through the fistula directly into the pulmonary veins and thereby into the greater circulation without having been arterialized. The larger the cross-section of the fistula and the more centrally located it is, the more blood is drawn into the pulmonary circulation and the poorer is the oxygen saturation of the peripheral blood. Much of the blood may pass through the shunt into the greater circulation. In such cases the arterial oxygen saturation is decreased. Values of 70 per cent are mentioned

in the reported cases.[5, 8, 23, 24] Cyanosis usually reaches a considerable degree. In spite of this, as signs of compensatory equilibrium, the arterial oxygen combining power and the arterial oxygen content become proportionately larger. Increased blood volume attributed to the increased cell volume with a normal quantity of plasma has been reported.[24] In these cases no enlargement and no change in the shape of the heart is found. This stands in contradiction to the arteriovenous aneurysm of the greater circulation where both the number of cells and the quantity of plasma are increased with normal hematocrit values, and the heart is frequently overloaded.[5] All authors have found normal circulation times and normal cardiac pressure relationships. The pressure in the lesser circulation is lowered by the short circuit. Enlargement of the heart after postoperative exclusion of the short circuit with elevation of pressure in the lesser circulation has been mentioned by Lindgren.[23]

The formation of a short circuit in the lesser circulation forms the basis for the symptoms and signs of arteriovenous aneurysm; cyanosis, dyspnea, secondary polycythemia, and pulmonary osteoarthropathy appear in these cases with stereotyped regularity. The extent of the symptoms and signs proceeds parallel with the size and position of the fistula; small fistulas make few or no symptoms, while larger fistulas produce symptoms so severe that the patients are completely incapable of work. Almost all of these patients complain of shortness of breath, tiredness and weakness. Neurologic signs are mentioned relatively frequently; these are attributed to cerebral involvement. An explanation for this cerebral involvement may be the tendency to thrombosis in polycythemic states or by emboli which arise from the mural thrombi of the pulmonary aneurysms. In some cases, simultaneous occurrence of cerebral aneurysm is found. Hemoptysis occurs in about one-sixth of the cases. Dullness over the thorax is demonstrable only in the case of aneurysms of considerable size. In over half of the cases, at the point of the thoracic wall closest to the aneurysm, a humming murmur synchronous with the heart can be heard; this increases markedly during systole and with inspiration. High-grade anemia, occurring after hemorrhages from telangiectases of the mucous membranes in Osler's disease, may mask the cyanosis and the polycythemia; these appear again, of course, if a hemorrhage-free interval occurs.

The clinical picture of arteriovenous fistula may remind one of the cyanotic type of congenital heart diseases. The normal size of the heart and the normal findings on auscultation, as well as the radiographic demonstration of the aneurysmal shadow, protect us from this error. Many fistulas of this kind have been misdiagnosed as congenital heart lesions. The normal heart findings may also misdirect one to the incorrect diagnosis of essential polycythemia. When all typical symptoms are present the clinical diagnosis produces no difficulties. In the case of small fistulas, essentially without symptoms, the diagnosis becomes much more difficult.

The radiographic diagnosis is essentially that of the differentiation of a round shadow of the lung. The following signs are characteristic for arteriovenous fistulas: round, ovoid or polycystic, delimited shadows in the lung parenchyma

which are connected with the hilus by broad, spirally coursing shadows. These stripe-like shadows represent the vascular connections with the hilus and were first described as characteristic in 1933 by Barnes.[6] With increasing size the round shadows usually develop a racemose form. In addition to the larger shadows, one frequently recognizes smaller nodules which correspond in their cross-section to the spirally running afferent and efferent vessels. These shadows are usually compact and sharply delineated against the surrounding lung tissue. If the aneurysm increases in size, it produces compression of the surrounding pulmonary tissue with atelectases, pneumonic thickenings, indurations and hemorrhage into the pulmonary tissue. Thus, the earlier sharply delimited roentgenographic shadow may appear to be indefinitely contoured or may not be able to be demonstrated at all roentgenologically. Pulsation of the round shadow has been found in only about a fourth of the cases. Lindgren[23] was the first to point to the changes in size with Valsalva's experiment. Calcium plaques in the wall of the aneurysm may produce a semilunar shadow. Tomography may aid in the diagnosis by the demonstration of the widened, spirally running vessels as well as the exact localization. Angiocardiography produces absolute proof. The afferent vessel, the aneurysm, and the efferent vessel can be represented full of contrast medium in rapidly repeated serial views. Diagnosis, however, is almost always possible without the additional help of contrast medium. Angiocardiography is especially conclusive in small nodules, in aneurysms which lie behind the heart, in round shadows lying close to the mediastinum, and for the discovery of additional nodules which do not visualize on the plain film. This is especially important in regard to the question of operability.

Surgical removal is the only therapy which can produce complete cure. While single nodules are ideally operable, multiple nodules are operable only if no more than two lobes of a lung must be removed. The most ideal method of operation is represented by excision of the aneurysm. The usual operation, however, has been lobectomy. Both methods of operation have led to complete relief of symptoms. It remains to be seen whether the method of only ligating the vessels is justified because of the incompletely excluded danger of rupture and the fact that the aneurysm may be fed by collateral vessels. The cases reported of Moyer and Ackermann[27] are interesting, in which, after the lung operation, telangiectases of the face and lips developed. If, in the presence of several arteriovenous fistulas each nodule is removed, enlargement of previously dormant fistulas may follow, making further operation necessary. Baer[4] reported a patient who had to be operated upon three times consecutively on this account. Shortly following the last operation, a new fistula appeared. Whether small symptomless fistulas should be operated upon is debatable. Usually one awaits the appearance of symptoms. In small fistulas this usually involves the risk of rupture. The tendency to thrombosis due to the presence of polycythemia is an important consideration. The prognosis is most unfavorable in those patients not operated upon because of the danger of rupture, which is considerable.[19, 31, 37]

Aneurysms of the peripheral branches of the pulmonary artery occur on the basis of an acquired injury of the vessel wall, and with the exception of the small

aneurysms in the walls of tuberculous cavities (Rasmussen aneurysms),[29] are very rare. In the case of tuberculosis, the Rasmussen aneurysms are found in 4 to 5 per cent of necropsies of patients dying of chronic pulmonary tuberculosis. Auerbach[3] reported 45 cases in 1114 autopsies of persons dying of chronic pulmonary tuberculosis.

The unusual occurrence of peripheral pulmonary aneurysms unassociated with tuberculosis has its basis preponderantly in the fact that these vascular changes almost always appear as a consequence of a basic disease with severe symptoms. Such diseases as chronic infections and polyarthritis may underlie the symptomatology. Radiologically they usually represent accidental findings. A diagnosis during life has been made in only a few cases. The vascular injury is usually associated with the embolic spread of mycotic material. The most frequent starting point is ulcerative endocarditis. A patent ductus arteriosus or a septal defect was found in about half of the cases.[20, 21, 22, 32] These malformations present a locus minoris resisteniae for endocarditic infection. Furthermore, by the formation of a left-to-right shunt, dissemination into the lesser circulation is possible. In addition, elevation of pressure in the lesser circulation may thereby subject the division points of the pulmonary vessels to an increased mechanical stress so that they form places of predilection at which bacterial emboli localize. Primary mycotic aneurysms are also possible without primary cardiovascular malformations.

Rheumatic vascular wall injuries comprise one large group of these rare cases. The chief stem and main branches of the pulmonary artery are almost regularly affected. Histologically these lesions give the picture of a mesopulmonitis.

Inflammatory changes in the neighborhood of the pulmonary arteries, in addition to mycotic and rheumatic infections, can lead to aneurysm formation. To this group belong the previously mentioned small aneurysms in the walls of tuberculous cavities, as they were first described by Rasmussen[29] in 1896. These are small, and usually reach only the size of a pea, and since they are obscured by the chronic tuberculous process, are not accessible to radiologic demonstration. Clausen,[11] in a case of chronic pneumonitis with abscess formation, described this as the cause for the appearance of a cherry-sized aneurysm.

Occasionally, in the literature are found reports concerning the traumatic origin of pulmonary aneurysms following shotgun injuries.[10, 25] These traumatic aneurysms can develop even years following the wound, increase in size, and finally lead to death by rupture.

The acquired peripheral pulmonary aneurysms are spherical, blood-filled structures in the pulmonary tissue; they are usually found in the region of the hilus as distinguished from the congenital forms. They are always formed at the division points of the arterial branches especially exposed to mechanical stress. The right side is affected more frequently than the left. The chief stem is involved in more than half of the cases, and it is more or less aneurysmally dilated. Usually these aneurysms are multiple and vary in size from 0.5 to 4 cm. in diameter. They are always unicameral. Sac-like projections, subdivisions of

hollow spaces standing in connection with one another, as well as dilated afferent and efferent vessels—as one sees these features in arteriovenous fistulas of the lung—are lacking. Radiologically, the round shadows close to the hilus are characteristic if they are arranged corresponding to the localization of vascular divisions. If they show intrinsic pulsation, diagnosis is relatively easy. As a rule, the smaller aneurysms of longer duration are filled by thrombi and pulsations are lacking. The roentgenographic picture is completely uncharacteristic. From the standpoint of differential diagnosis, the large group of tumors which occur about the hilus, round tuberculous foci, and cysts must be considered.

In spite of their rarity, it is important in the presence of atypical round shadows lying close to the hilus to think of the possibility of the existence of pulmonary aneurysms. The history of chronic infection and polyarthritis, the existence of malformations of the heart, of the large vessels, or of an aneurysmal dilatation of a pulmonary stem, and the appearance of hemoptysis helps further in diagnosis. A circumscribed vascular murmur would be evidence of the aneurysmal nature of a round focus. However, on account of the position close to the hilus and the usually present murmurs found upon auscultation of the heart, diagnosis is very difficult. The heart is enlarged in all of these cases. Further difficulties in diagnosis are produced if the surrounding lung tissue is altered by inflammatory changes due to injuries to the vessel walls. Thus, there may be fresh areas of pneumonitis, chronic indurations, abscess formation, or septic infarcts. On account of the same density of shadow of the vessels and of the infiltrated lung tissue, delimitation of the aneurysm may no more be possible by radiography. This occurs more frequently with acquired aneurysms than with the congenital ones.

The development of peripheral aneurysms can be followed by means of radiography. Weise[36] reported a case in which lively pulsating nodules became larger and thicker within two years. Peripheral aneurysms are not insignificant structures, since fatal ruptures are frequent (Arel,[1] Castex,[10] Clausen,[11] Marble,[25] Sachs[32]). Operation is usually impossible because of the severity of the underlying disease. Arel[1] performed an operation in which an *orange-sized* nodule was removed from the right lower lobe. This had been diagnosed an echinococcal cyst. At operation, however, two *orange-sized* aneurysms were found.

Hückstädt[18] described a case of peripheral aneurysm of the pulmonary artery. This patient had a loud, systolic murmur over the lower right lung field. Roentgenography showed a bucket-form dense shadow lying anteriorly in the midportion of the lower lung field over which and toward the hilus was an almond-sized shadow. The synthesis of the roentgenographic findings with the presence of a murmur led to the correct diagnosis. The band-like shadows leading to the hilus were regarded as congested branches of the pulmonary artery, and the round shadow as an aneurysm. Cardiac failure and cerebral embolism resulted in death. Autopsy showed a simple aneurysm of a middle-sized branch of the pulmonary artery.

CASE REPORTS

Case 1. J. R. K., a 47 year old man, had a miniature chest roentgenogram made by the Red Cross Mobile Unit in August 1951. A larger chest roentgenogram was requested because of *cloudy appearance* about the left hilus. His only symptom was a cough starting six months previously following a cold. The cough had been productive of clear, nonbloody sputum especially in the morning on arising. He had had chronic sinusitis with postnasal discharge for a year. He also had a vague sensation of pain in the left chest.

Physical examination showed a well developed, slightly built man, weighing 137 pounds. The pulse was 66; blood pressure was 158/90. There were no heart murmurs and no abnormal sounds over the pulmonary fields. Laboratory examination demonstrated a hemoglobin level of 14.3 Gm. per 100 cc. The red cell count was 4.31 million per cu. mm. and the total leukocytes numbered 6,800 per cu. mm. The urinalysis was negative. Roentgenographic examination of the chest showed the thorax to be symmetrical and the diaphragm to be smooth; the cardiovascular shadows were not remarkable, except that the hilar densities were prominent especially on the left (fig. 1). The right lateral projection showed a density in the left pulmonary hilus which could be clearly separated from the aorta (fig. 2). There was no visible pulsation of this mass on fluoroscopy. Bronchoscopy on Nov. 14, 1951 was completely negative; a bronchial sponge biopsy was negative.

On November 15, a left thoracotomy was done removing the sixth rib subperiosteally. No lesions was found in the lung. The pulmonary artery on the left side was as large as 6 cm. in diameter in one circumscribed area, bulging in its first portion. It pulsated markedly with a definite thrill. This represented an aneurysm of the trunk of the pulmonary artery. The vessel was dissected out, and a strip of cellophane was wrapped around it. The pleura was then closed over the cellophane. The wound was closed with interrupted sutures following the institution of waterseal suction drainage. The postoperative course was entirely uneventful, and the patient has remained asymptomatic up to the present time (Nov. 1, 1953).

Case 2. L. M., a 64 year old man, was admitted to the hospital on Feb. 9, 1952 with the complaint of chills and fever with generalized aching for the preceding two days. For the past year he had noted increasing dyspnea and a cough productive of yellowish-white sputum, especially in the morning. During the past month this sputum had become occasionally rusty with flecks of blood. He had had dull pain along the right costal margin which was relieved by bending over and putting pressure over the area. The pain lasted up to three-quarters of an hour in attacks. The last episode of pain had occurred three weeks before. He had had edema of the ankles in the past, and occasional dizziness with exercise. He worked until four months previously at which time he was retired. He had lost no weight in the preceding year.

Examination disclosed a well-developed man, weighing 185 pounds. The pulse was 70; the blood pressure was 120/70. The temperature was 102 F. His skin was moist and warm. The heart sounds were normal, and there were no murmurs. Breath sounds were diminished over the right lung base with many coarse rales. Crepitant rales were heard throughout the left lung base. The remainder of the physical examination was normal. Urinalysis was negative. Blood examination showed hemoglobin 14.3 Gm. per 100 cc., red cell count 4,660,000 per cu. mm. and white cell count 8,100 per cu. mm. Sedimentation rate was 25 mm. per hour (Westergren).

Roentgenographic examination demonstrated a mass in the right pulmonary hilus which did not pulsate (fig. 3). The heart was not enlarged in the transverse diameter, but there was prominence of the pulmonary artery segment. The ascending aorta was widened. Some right ventricular hypertrophy was present. The circumscribed density in the right hilum was apparently not due to lymph node enlargement. There was evidence of some collapse of the middle lobe as evidenced by linear accentuation along the right cardiac border. The hilar density was somewhat increased on the left. The intermediate and peripheral zones of the lung fields were clear. The heart was somewhat enlarged, especially to the left. The retrocardiac space was lessened by right auricular enlargement (fig. 4). It was thought that these

FIG. 1. Case 1. J. R. K. Central type aneurysm of pulmonary artery. Anteroposterior view shows left hilar density.

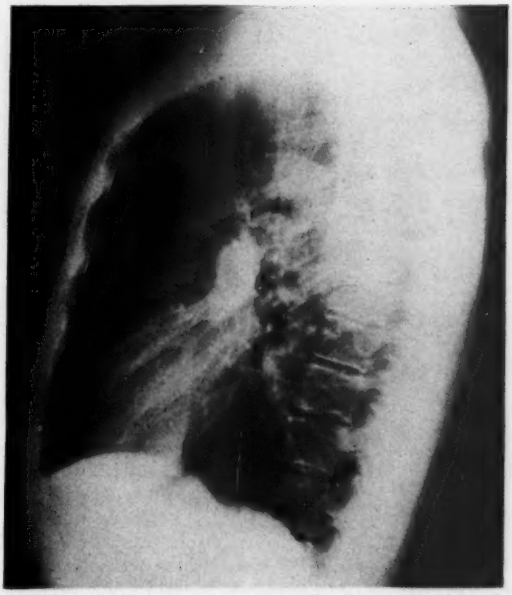

FIG. 2. Case 1. J. R. K. Central type aneurysm of pulmonary artery. Lateral view shows density in left pulmonary hilus clearly separated from the aorta. Mass did not pulsate on fluoroscopy.

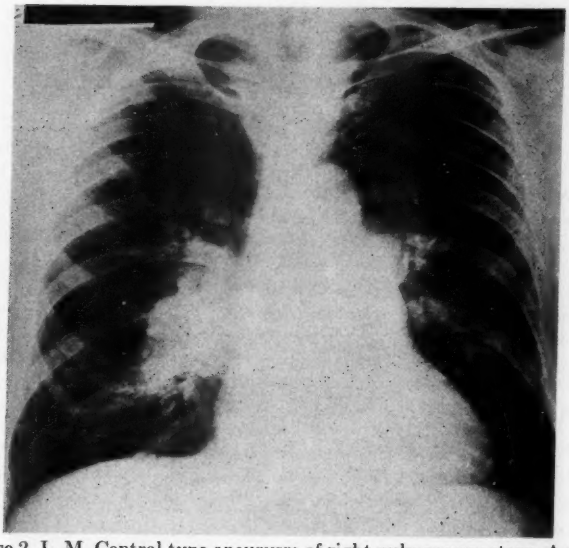

FIG. 3. Case 2. L. M. Central type aneurysm of right pulmonary artery. Anteroposterior view shows a large mass in right pulmonary hilus. Some collapse of middle lobe. Heart somewhat enlarged to left.

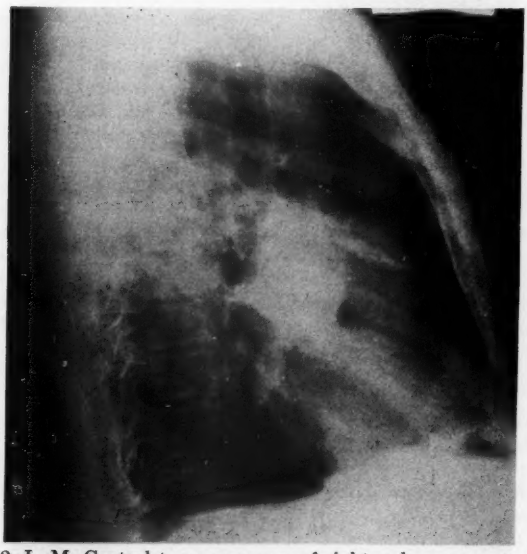

FIG. 4. Case 2. L. M. Central type aneurysm of right pulmonary artery. Lateral view shows a mass in the right pulmonary hilum. Retrocardiac space lessened by right auricular enlargement.

findings were due in part to pulmonary congestion. However, the radiologic impression was that these findings represented a carcinoma of the right lung.

Bronchoscopy was done under local anesthesia on Feb. 13, 1952. The carina was much widened, but not blunted. On the right, beyond the upper lobe orifice there was marked narrowing of the bronchus with bulging of the medial wall. Some granulation tissue was present around the middle lobe orifice and around the dorsal division of the lower lobe orifice, but no ulcerating tumor could be seen. Tissue for biopsy was taken from these regions and was subsequently reported to show only chronic inflammatory changes.

Electrocardiogram showed a rate of 90, rhythm regular, left axis deviation. T-wave showed low amplitude in all leads. The impression was diffuse myocardial damage with a hypertensive configuration.

Right thoracotomy was performed on Feb. 16, 1952, removing the sixth rib sub-periosteally. There was a large mass in the hilum of the right lung. This was palpated and found to pulsate. It represented a large aneurysm of the right pulmonary artery which measured 6 cm. in its greatest diameter. The aneurysm extended peripherally to the bifurcation of the vessel into the upper and lower branches. The pulmonary vein was somewhat dilated by compression. It was diffusely enlarged and there was no weakening of the vein wall. The right stem bronchus was stretched over the dilated sac of the aneurysm. It was not feasible to wrap the aneurysm because of fixation, and it was not believed that pulmonary resection was indicated. The chest was closed in layers following the institution of closed intercostal waterseal drainage. The postoperative course was uneventful and the patient has remained in his preoperative status with recurring bouts of fever and pneumonitis up to the present time (Nov. 1, 1953).

Comment. Both of these cases represent aneurysms of the pulmonary artery of the central type. It was possible to mobilize the aneurysm in the first case and wrap it with reactive cellophane. The aneurysm in the second case was so large and so adherent to the surrounding structures that any definitive therapy was impossible. The production of a benign bronchial stricture by external pressure of an aneurysm with resulting chronic obstructive pneumonitis was noted.

The cause of death from a central aneurysm of the pulmonary artery is most commonly heart failure or rupture of the aneurysm. In such a series of 36 central aneurysms, Deterling[12] found 4 cases in which death occurred because of rupture. These ruptures occurred into the pericardial sac and into a bronchus. The paucity of cases makes any definite conclusions regarding choice of procedure impossible.

Case 3. L. L. L., a 42 year old white farmer, entered the hospital on April 22, 1951. For the past 15 years he had been having frequent severe nosebleeds which were attributed to sinusitis. These episodes became so severe that at one time he was given treatment for anemia. Approximately 10 years before he had had an episode of hematemesis and melena. The melena lasted for 20 days. The following year he began to lose weight, cough, and have blood-streaked sputum, and was admitted to a tuberculosis sanatorium where positive sputum was said to have been obtained at one time. He was released after 22 months of treatment and was able to farm successfully. In 1944 he again had tarry stools but no hematemesis. This episode lasted one week. In 1948 he had another four day episode of tarry stools. In the spring of 1948 he first noticed the onset of wheezing, cough, dyspnea, and occasional sneezing bouts. These episodes were diagnosed as asthma and treated effectively with adrenalin. They had increased in severity and frequency in the past two years to the extent that they had interfered with his holding a job. Three weeks prior to admission to the hospital he consulted his local physician who, after roentgenographic examination of his chest,

told him that he had a pulmonary lesion and referred him to this hospital. System review revealed only bouts of weakness associated with the occurrence of tarry stools. His mother had frequent nosebleeds to the point of unconsciousness and occasional episodes of bleeding from the conjunctivae. No familial history of skin telangiectases was obtainable.

Physical examination revealed a thin, white man in no acute distress. There were hemorrhagic crusts in both nares, and scattered over the pharynx, hard palate, and lips were many small, bluish areas which blanched on pressure. A few of these were also noted on the trunk and fingers. The ocular fundi were normal. The chest was thin with expiratory wheezes heard throughout both lung fields. There was a systolic bruit best heard in the supine position in the midaxillary line over the seventh interspace. This was separate and distinct from the heart sounds. Blood pressure was 115/60; the abdomen was negative. Bronchoscopic examination revealed no telangiectasia in the visible tracheobronchial tree.

Laboratory examination demonstrated a hemoglobin level of 10.5 Gm. per 100 cc., red cell count of 4,300,000 per cu. mm. and white cell count of 6,000 per cu. mm. with a normal differential distribution. The bleeding time was 1 minute 5 seconds; the clotting time was 4 minutes 20 seconds; the hematocrit was 39. Serological tests for syphilis were negative. Stools were negative for occult blood on 3 occasions. Sputum was negative 3 times for acid-fast bacilli by smear and cultures. The arterial oxygen saturation was 85 per cent. The plasma volume was 55.5 cc. per kg. Blood volume was 94.7 cc. per Kg. Urinalysis was negative on a voided specimen, and on a specimen obtained by ureteral catheterization.

Roentgenographic examination of the chest (fig. 5) demonstrated evidence of old apparently arrested tuberculosis at both apices, more marked on the right than on the left. In addition there was a smoothly outlined homogeneous density, ovoid in shape, in the left lower lung field which was demonstrated to be in the parenchyma and not attached to the pleura. It was very questionable whether this mass pulsated or not. There was another very discrete

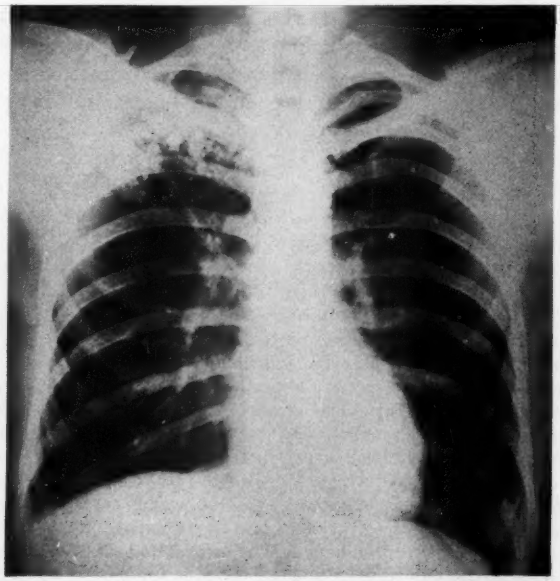

FIG. 5. Case 3. L. L. L. Peripheral arteriovenous fistula, left lower lobe. Anteroposterior view shows old fibrotic tuberculosis both apices. Smoothly outlined, ovoid, homogeneous density in the left lower lung field. Another very discrete, rounded area of density, 1.5 to 2 cm. in its greatest dimension in the right lower lung field.

FIG. 6. Case 3. L. L. L. Peripheral arteriovenous fistula, left lower lobe. Lateral view shows anterior location of mass.

rounded area of density 1½ to 2 cm. in its greatest dimension which was detected in the right lower lung field. It was demonstrated to lie posterior to the mid-coronal plane and therefore apparently in the lower lobe (fig. 6). Angiocardiography was done in an attempt to fill these lesions, but it was unsuccessful. Retrograde urograms were made and were seen to be within normal limits. Barium enema and gastrointestinal series were both negative. It was believed that the pulmonary lesions represented arteriovenous fistulas of the lung associated with other manifestations of hereditary hemorrhagic telangiectases.

On May 26, 1951 a left thoracotomy was done, and a large pulsating hemangioma and several smaller ones were found in the left lower lobe. A lobectomy of the left lower lobe was done utilizing the individual ligation technic. The chest wall was closed following the institution of waterseal intercostal closed drainage. His postoperative course was entirely uneventful.

The report on the surgical specimen was peripheral arteriovenous fistulas of the left lower lobe. His condition has remained good to the present time and there has been no evidence of development of further pulmonary arteriovenous fistulas. The lesion in the right lower lobe has remained unchanged, two and one-half years later.

Case 4. R. H. T., a 64 year old white man, had been admitted to this hospital in 1948 at which time he had fainted and struck his head on a curb. An epidural hematoma was surgically removed from the right temporal area at that time. During that admission a chest roentgenogram revealed a lesion in the right lung of the round coin type. He was admitted again on July 5, 1951 following a miniature roentgenogram made by the mobile unit, for the evaluation of this lesion. He had no cough and apparently had been in good health.

Examination disclosed a blood pressure of 130/80. Clinical evidences of moderate emphysema were present. A to-and-fro murmur was heard over the right nipple area. Laboratory studies were all within normal limits. The electrocardiogram was within normal limits.

Roentgenographic examination showed (fig. 7) a coarsely lobulated mass measuring about 4 cm. in diameter in the right midlung field intimately associated with large vessels coursing downwards from the right hilum. Oblique projection localized the lesion to the anterior portion of the chest. The cardiac shadow appeared normal in configuration and size.

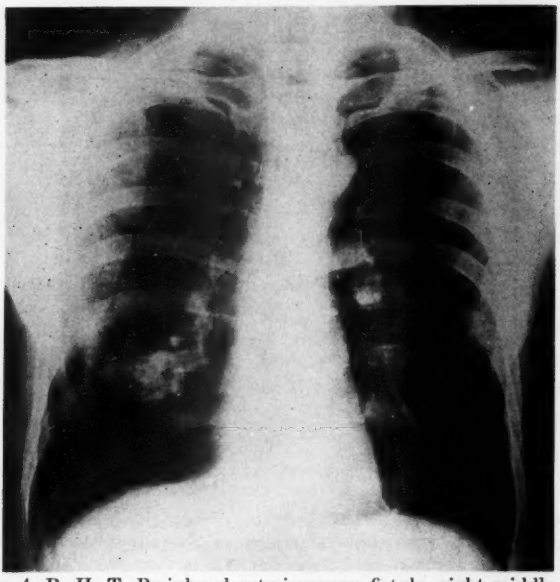

FIG. 7. Case 4. R. H. T. Peripheral arteriovenous fistula, right middle lobe. Anteroposterior view shows a coarsely lobulated mass 4 cm. in diameter intimately associated with large vessels coursing downwards from the right hilum. Cardiac shadow normal. Healed inflammatory disease, right apex. Moderate bilateral emphysema.

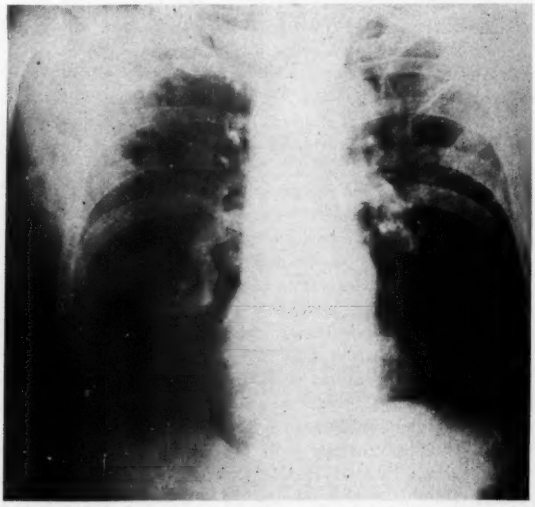

FIG. 8. Case 4. R. H. T. Peripheral arteriovenous fistula. Anteroposterior view, angiocardiogram. Confirms finding in figure 7.

The lung fields were otherwise clear except for discrete calcific densities in the lower left lung field and left hilum, and also some fibrous-appearing strand-like densities in the right apex at the first and second interspaces. Both bases showed increased translucency and flattening and irregularity of both leaves of the diaphragm. The impression from this examination was an arteriovenous aneurysm in the right midlung field, moderate bilateral emphysema, and healed inflammatory disease of the right apex. Angiocardiography was done, and the impressions derived from the other roentgenographic examinations were confirmed (fig. 8).

Because of his age and emphysema, surgical therapy was not recommended at the time of examination.

Comment. These last 2 cases of pulmonary arteriovenous fistulas are very typical. Case 3 represents multiple pulmonary arteriovenous fistulas occurring in an individual with other manifestations of telangiectasia hereditaria hemorrhagica (Osler's disease).

A significant incidence of serious or even lethal complications in untreated cases (hemoptysis, hemothorax, bacterial endarteritis, brain abscess, thrombosis associated with polycythemia) leads us to recommend surgical measures except in persons of advanced age, in those seriously ill of unrelated disease, and in patients who have widely distributed lesions. Surgical resection should be only as extensive as necessary to extirpate the lesion. Simple ligation of the supplying vessels is not recommended because of numerous collateral vessels.

SUMMARY

1. Two cases of central aneurysm of the pulmonary artery and 2 cases of pulmonary arteriovenous fistula are presented.

2. Central aneurysms of the pulmonary artery are uncommon lesions. They produce death by heart failure and less commonly by rupture. From the standpoint of differential diagnosis they must be distinguished from mediastinal nonvascular masses. Recognition of associated cardiac lesions is important.

3. Pulmonary arteriovenous fistulas are more common than central aneurysms and must be distinguished from round, parenchymal pulmonary lesions.

4. Surgical intervention is always recommended except in cases with numerous, widely distributed lesions or complicating disease.

5. Simple aneurysm of the branches of the pulmonary artery are rare lesions. They are discussed briefly.

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A TRANSPERIOSTEAL INTERCOSTAL INCISION*

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With thoracotomy now commonplace and resulting in seemingly satisfactory healing, some comment is in order before offering a new technic. The very fact that so many chests now are being opened for a wide variety of reasons justifies critical reappraisal of conventional methods, particularly by the thoracic surgeon whose emphasis shifts to jealous preservation of all possible function. Any details decreasing postoperative pain obviously are important in reducing complications through easier coughing and through reduction of narcotics. They also are important, if they minimize pain or any late deformity and scar, in promoting better acceptance by the public and by the profession of elective procedures such as removal of tissue from the lung under direct vision for biopsy study. Exploratory thoracotomy for this purpose is a procedure which has been sadly neglected in many areas.

Advocates of intercostal incision not only claim less external deformity than often results from rib resection, but also claim less postoperative pain, a point difficult to demonstrate with statistically valid observations. The present trend toward intercostal incisions undoubtedly has been influenced by numerous patients who calmly accept the prospect of the most major thoracic surgery, but hasten to inquire with alarm if any ribs are to be removed.

Precise surgeons note, however, that with the usual intercostal incision an accurate and functional reconstitution of the frayed intercostal muscle fibers at the conclusion of the operation is a practical impossibility, since the sutures strip out along the direction of the muscle fibers, which are friable at best. Placement of numerous interrupted sutures in this layer not only seems futile, but creates some risk of postoperative bleeding from intercostal vessels. On at least three occasions I have been asked for consultations on patients whose bleeding seemed to arise from such a source. There also is risk of producing protracted pain by catching nerve filaments in the sutures. Some therefore have abandoned the suture of this layer entirely, relying upon prompt expansion of the underlying lung, and upon careful closure of the overlying fascial layers and skin to maintain closure of the chest wall until a thin plate of scar reorganizes an intercostal layer.

TECHNIC

During the past year the author has used for routine thoracotomy an incision which has not been found previously described, but which appears to provide a simple solution to the problems discussed above. It is essentially intercostal

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but maintains the intercostal muscles as functioning units with their normal nerve and blood supply. It also minimizes bleeding, by entry through the chest wall by way of the upper rim of the periosteum of the rib below, and retaining this narrow strip of periosteum to hold the intercostal muscles together for the closure (fig. 1). This reduction of irritating suture material to a minimum not only aids in painless healing, but may be found of particular value in cases such as coarctation where the collateral vessels impose a troublesome problem

The periosteum of the rib below the selected interspace is incised along a line

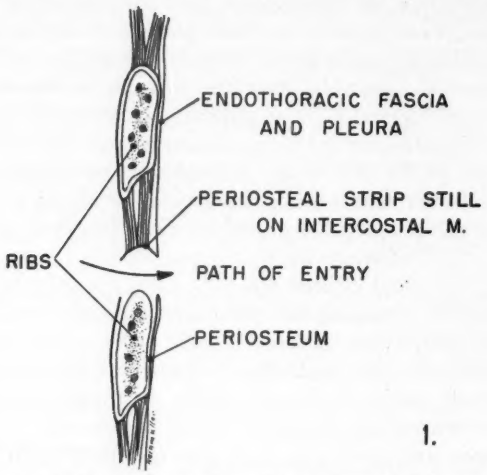

FIG. 1. Method of making incision through periosteum

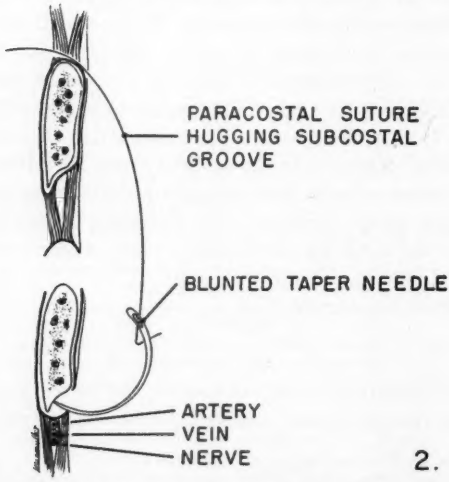

FIG. 2. Method of placing paracostal sutures for wound closure

one fourth of the distance from its upper edge. The lower or principal area of periosteum is at all times undisturbed, but the upper flap is freed in the usual manner by passing a periosteal elevator along the bone distally to free the upper or cephalad edge. The interspace then may be spread with a finger while the pleural cavity is entered through the periosteal bed without attempting to free it from the inner surface of the rib. As with any thoracotomy performed in the lateral position, it is helpful to have the rib cage spread slightly by elevation of a kidney lift just above the iliac crest, or placement of a tight roll under the patient at this level.

In closing the chest, the strain is taken by no. 00 chromic catgut paracostal sutures which are passed with a blunted taper point (kidney) needle with particular care to slide up against the lower edge of the rib below, in such a fashion as to avoid the nerve filaments and vessels by staying within the periosteal envelope (fig. 2). Since the subcostal groove is tilted inward, this may be facilitated by approaching it from within the chest, or in other words, starting the suture from the outside by going over the rib above the interspace entered, and coming out below.

Fortunately, it is found the intercostal muscles tend to curl in, so that if the outer lip of the little periosteal trough is caught up with a light suture to the adventitia on the rib, the trough tends to ride back upon the rib like a saddle (fig. 3). No effort is made to make this layer air tight.

Application of the incision to the anterior interspaces is important, because of the utility of the supine position in certain cases and because this is the site for the thoracotomy we use to obtain tissue for biopsy. It is made somewhat more difficult where the perichondrium does not strip away so readily and where the ribs cannot be drawn together sufficiently to return full contact of the layer to the cartilage below. However, the same principles still hold.

With the incision here described a reduction in the requirements for post-

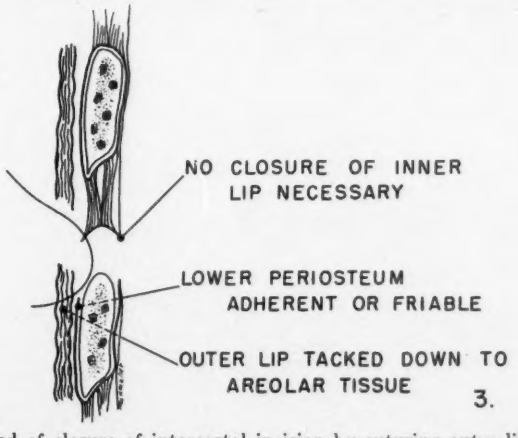

FIG. 3. Method of closure of intercostal incision by suturing outer lips of periosteal through to the adventitia of the rib.

operative narcotics has been noted, but this matter is inevitably influenced by well meaning nurses who long since have come to believe that after major surgery use of certain amounts of narcotics is to be expected, discounting the patient as either being unduly brave or cantankerous. We have noted very real evidence of local pain reduction in the fact that patients formerly so concerned with their chest that other distresses were ignored now often mention other areas such as the back, or the sites of injections.

CONCLUSIONS

A transperiosteal intercostal incision is presented for routine thoracotomy, which has been found rapid, and an efficient means of minimizing postoperative bleeding, pain and deformity or loss of function.

MASSIVE SOFT TISSUE INJURY TREATED WITH EARLY SPLIT THICKNESS SKIN GRAFTS

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INTRODUCTION

Massive soft tissue injury has become a problem of increasing frequency in this modern era. When the clinician is confronted with a patient having debilitating injuries of this type, the efforts of his therapy are concerned primarily with obtaining a satisfactory functional result. His most immediate problem, with reference to the wound, is the preservation of those structures which can be returned to a functional state. To do this, it is important to protect major nerves, vessels, and muscle groups which may be exposed.

It is the purpose of this paper to present evidence of the utility of early split thickness skin grafting associated with adequate early debridement in protecting tissues and preserving function. The typical cases will be presented with illustrations. Although these cases are examples of major trauma as a result of war wounds, the principles applied in their treatment are in many ways applicable to civilian practice. In these cases, there was extensive exposure and destruction of large muscle groups and their associated vessels and nerves. By early and extensive debridement followed by skin grafting, secondary infection was prevented, and scar tissue formation was held to a minimum. This in turn made early muscle rehabilitation through physical therapy possible. Split thickness skin grafting may not be considered the final corrective measure. However, it permits those tissues which remain to become functional in many instances at an early date. If tissue remains exposed for a period longer than 10 days to 2 weeks, it will degenerate to the point that its functional return may be impaired and secondary infection may develop. Such an area commonly becomes overgrown with scarred granulation tissue. Early coverage of a wound with split thickness skin graft may prevent these complications. Corrective surgery, such as the use of a pedicle flap, can be considered later.

The cases presented are casualties of the Korean War. They are examples of massive soft tissue injury which were treated with early and adequate debridement and split thickness skin grafts. It is the author's opinion that their functional return was greatly augmented by this treatment. In the cases presented, all grafts were taken with a Padgett dermatome. The thickness of the graft was at the discretion of the operator. As a general rule, those areas in which the blood supply was less abundant were grafted with thin grafts, whereas those areas with more rich blood supply were grafted with thicker grafts. All grafts varied from 0.015 to 0.020 inch. Grafts were held in place with interrupted sutures and with pressure dressings. No special procedure in grafting was followed, but strict

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hemostasis and careful immobilization were emphasized. In the cases presented, all questionably viable tissue was removed by extensive debridement before grafting. Patients routinely received penicillin and streptomycin postoperatively.

During a service of 12 months in Japan, the author treated approximately 500 cases of traumatic wounds, many with early skin grafting. Two of these with massive wounds received during the Korean War are chosen for detailed presentation below.

CASE REPORTS

Case 1. A white man, aged 22, sustained an explosion type injury as a result of enemy action in Korea. He was given emergency surgical treatment and was evacuated to a general hospital in Japan within three days. On examination, the inner aspects of both thighs were found denuded of skin and subcutaneous tissue from the inguinal ligaments to the knees. An area comprising 50 per cent of the diameters of the thighs was involved (fig. 1). On the right side, the femoral artery lay exposed, and on the left side the sciatic nerve was exposed. The major muscle groups of the anterior and medial aspects of both thighs were shredded and partially destroyed. The patient's general condition was satisfactory and his wounds were reasonably clean. He was taken immediately to the operating room and under a general anesthetic his wounds were further widely debrided. Four drums of split thickness skin were removed from the lower chest and abdomen. Portions of the wounds were secondarily closed, and the remaining portions were skin grafted. Split thickness skin was placed directly over the femoral artery on the right side and directly upon the exposed muscle groups. The grafts were held in place by multiple interrupted sutures and by pressure dressings (fig. 2). Immobilization was maintained by bilateral long leg splints. The first dressing was changed at the end of 14 days (fig. 3), at which time the grafts had taken almost 100 per cent. Shortly thereafter the patient was started on active physical therapy, and within 20 days of the time of injury he was able to stand and to walk with the aid of a cane.

Case 2. A white man, aged 22, sustained wounds of the right lower extremity and thigh as a result of enemy action in Korea. His wounds were initially debrided at a mobile army

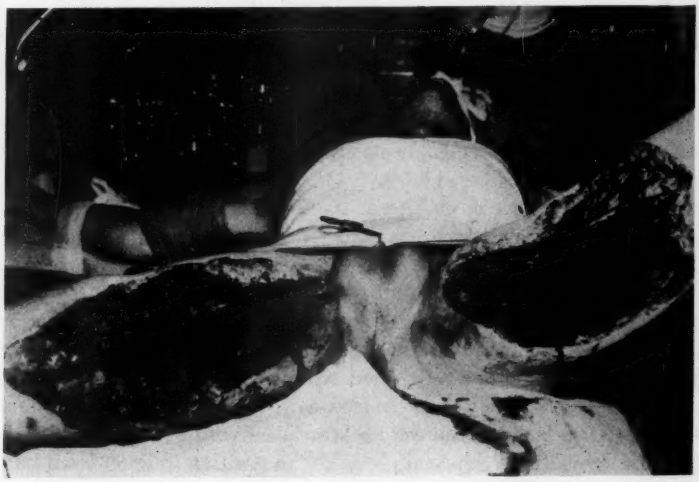

FIG. 1. Wounds (case 1) at time of operation. The right femoral artery is denuded, and the left sciatic nerve lies in the clotted region near the posterior portion of the wound.

FIG. 2. Grafted areas (case 1) immediately after operation involving partial secondary closure of wounds and split-thickness skin grafting of the remaining defects. Split-thickness skin grafts have been placed directly over the right femoral artery. The previously exposed left sciatic nerve has been transplanted into viable muscle with skin grafts over all.

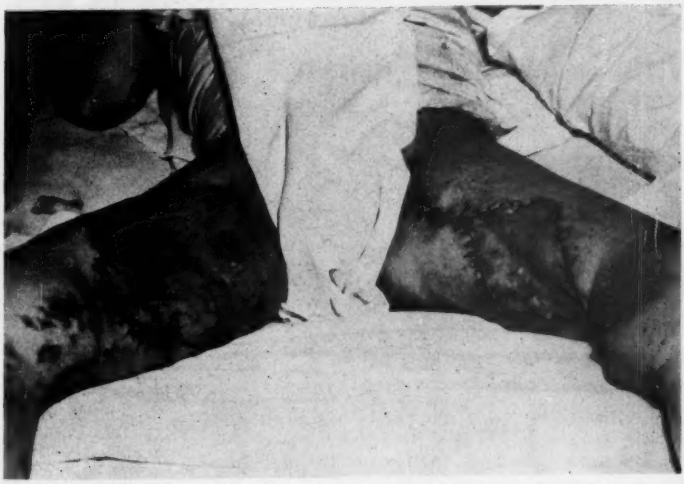

FIG. 3. Grafted areas (case 1) 14 days after skin grafting at the time of the first dressing change. The satisfactory take of the grafts is demonstrated. Active muscle re-education by means of physical therapy was begun on this date.

surgical hospital, and the right leg was amputated just below the knee. The patient arrived at a general hospital in Japan eight days later. On examination, the wounds of the right thigh were found reasonably clean. An area from the greater trochanter to the lateral aspect of the knee was denuded of skin and subcutaneous tissue. Approximately 40 per cent of the thigh was involved (fig. 4). Major muscle groups were exposed. The site of the amputation

FIG. 4. Wounds of right thigh and amputation stump (case 2) at the time of operation, eight days from the time of injury.

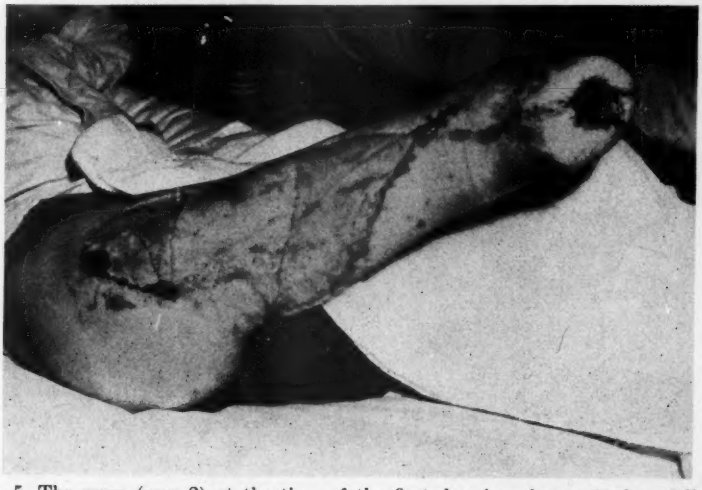

FIG. 5. The same (case 2) at the time of the first dressing change 14 days following operation.

was also reasonably clean. The patient was taken to the operating room and his wounds were again debrided. Three drums of split thickness skin grafts were removed from the abdomen and from the left thigh. These were placed over the denuded area directly upon muscle, and were held in place by multiple interrupted sutures and by pressure dressings. The right thigh was immobilized with a plaster splint. The first dressing was changed in 14 days, at which time an almost complete take of skin was noted (fig. 5). The patient was started on active physical therapy and quadriceps muscle exercises to prevent muscle wasting of the remaining portion of the right leg.

DISCUSSION

The treatment of this type of patient may be broken into several intermediate steps. Only portions of these will be discussed in detail. The first step is, of course, the immediate treatment of the patient with reference to shock and hemorrhage. This will not be discussed further.

As early as is practicable, the patient is taken to the operating room where his wounds are primarily debrided and further evaluated. It is at this stage that clinical judgment becomes of paramount importance. If the presenting wound has been massively contaminated, the surgeon may be content with extensive debridement and immobilization. He may elect not to attempt primary skin grafting at that time. This is almost universally true with war wounds, which many times have unusual degrees of contamination and considerable devitalized tissue which may not be readily apparent. The cases presented fall within this category. On the other hand, many such injuries suffered in civilian life are relatively clean at the time of initial surgery. Therefore, it may be the judgment of the surgeon to graft primarily. It is suggested that since skin is generally so readily available and removed with relative ease, primary grafting may be feasible in the vast majority of reasonably clean wounds.

The importance of wide and extensive debridement again should be re-emphasized. Those tissues (skin, subcutaneous tissue, and muscle) which are questionably viable at operation are essentially nonviable from a functional standpoint in my experience. They invariably will be replaced or bound down by scar tissue. This may be prevented, however, if all such questionable tissues are widely excised and primary split thickness skin grafts are applied.

In many instances major vessels will be exposed. Their smaller branches are sometimes avulsed and bleeding. These branches should be carefully ligated with no. 00000 or no. 000000 black silk. Every effort should be made to preserve any major vessels in a functional condition. If a vessel has been lacerated it should be debrided and resutured if possible, but if there is any doubt as to the viability of the edges to be approximated, resection with reanastomosis should be done. If a major vessel is left exposed, even though it be repaired and demonstrated to be patent at the time of initial operation, it will commonly slough and perforate, necessitating ligation. If split thickness skin grafting is not feasible at the time of initial operation, an attempt should be made to bury the vessel within or beneath viable surrounding muscle tissue. When the wound is reasonably clean, split thickness skin grafts can be placed directly over the exposed vessel, as in case 1, and may prevent complications.

When major nerves are exposed, as was the sciatic nerve in case 1, it is imperative that its surrounding tissue be viable. If the nerve remains surrounded by blood clot and nonviable tissue, it may slough. It is important that the operator debride widely about the exposed nerve, and if possible transplant it into viable muscle. When a nerve has been lacerated, the surgeon should attempt to determine before operation if it is totally damaged or only partially functional. If partially functional, he may attempt to reapproximate that portion which has

been lacerated. This again depends upon his surgical judgment for that given case. If the nerve has been completely severed, and if the wound is contaminated, the treatment of choice in most instances is to identify both cut ends and tie them with stainless steel wire ligatures. The nerve should then be buried beneath viable muscle, with the thought in mind of returning and anastomosing the nerve at a later date. By early wide debridement and careful removal of devitalized tissue, such complications as secondary infection and scar tissue formation about the nerve in question may be prevented.

War wounds frequently have complications not commonly seen in civilian wounds. They are the result of multiple penetrating fragments and unusual contamination. In wounds of this type, it is possible in many instances to graft denuded areas between the points of perforation at a fairly early period, within 3 to 10 days. It is not necessary or desirable to postpone grafting of the flat intervening surfaces even though the puncture wounds are infected and draining. The perforation points are left exposed, and are drained into the pressure dressing applied for the skin graft. This may considerably cut the time that a wound remains exposed and may permit earlier functional re-education.

SUMMARY AND CONCLUSIONS

There seems to be a correlation between the time of coverage of massive soft tissue wounds and the eventual satisfactory return of function in the part involved. If a wound is covered early, and functional re-education begun, as in case 1, the degree of functional return is greatly augmented.

After early extensive debridement many wounds can be primarily skin grafted.

By early extensive debridement, a wound not primarily skin grafted may be ready for grafting within a short time.

Extensive debridement and early split thickness skin grafting may prevent the loss of major nerves and vessels.

When a wound is adequately debrided, secondary infection and excessive scar tissue formation may be avoided. Primary skin grafting of reasonably clean wounds may be feasible in more instances than has been heretofore recognized.

Two cases of extensive dirty war wounds which were treated with wide debridement and early split thickness skin grafting have been reported. Because of this treatment these patients had an early return of function.

The author is indebted to Colonel V. A. Jeffress, M.C., U. S. A., for supervision and advice in the care of these patients, and to Dr. Henry N. Harkins, Professor of Surgery, University of Washington, for help in preparing this paper.

CLINICAL EXPERIENCE WITH HYPOTENSIVE ANESTHESIA

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The use of high spinal anesthesia is not new. The hazards and accidents that have been reported following this type of anesthesia have placed the procedure in a category of questionable value. More recently, because of the work of Gillies,[8] the problem of high spinal anesthesia as a method of producing the hypotensive state has been subjected to re-examination.

Two outstanding purposes are served by the use of hypotension during a surgical operation. First, the surgeon is provided with an operative field that is relatively free from bleeding. This facilitates the surgery and shortens the operative time. Second, blood is conserved for the patient, eliminating the necessity for replacement. Are these reasons sufficient to justify hypotensive anesthesia in the face of efficient blood replacement programs? This is a controversial issue. What are the hazards of hypotension in contrast to the hazards of blood replacement therapy? The answer is not known. It is believed that transfusion reactions are more likely to occur in patients who receive multiple transfusions.

Successful use of high spinal anesthesia to effect the hypotensive state embodies several important considerations. First, motor and sensory paralysis must be carried to the fifth thoracic segment with diffusion to the first thoracic segment in sufficient concentration to paralyze the sympathetic outflow. Second, no vasopressor drug is used. Third—and of utmost importance—is effective and adequate oxygenation of the patient. Hypoxia does not occur in the well oxygenated patient during hypotension because there is no interference with utilization of oxygen by the tissue cells. Fourth, efficient venous return must be maintained. Gillies[9] expressed the opinion that renal or hepatic failure will not follow hypotensive spinal anesthesia if the patient is well oxygenated during the operation and afterwards while the blood pressure is returning to normal levels. He also stated that a low blood pressure due to vasodilatation in the presence of a normal blood volume is less dangerous than a high blood pressure associated with vasoconstriction and a reduced blood volume.

The Trendelenburg position must be utilized to insure adequate venous return of blood to the heart. Enderby[6] stated that hypotension is a well recognized feature of spinal anesthesia and need not be feared if steps are taken to prevent its harmful effect by correct posture of the patient. Maxson[10] was emphatic in his opinion that in total anesthesia, either deliberate or accidental, the Trendelenburg posture must be used to prevent fatal cerebral and cardiac anemia. This posture is convenient in low abdominal surgery where it is employed to advantage to drain blood away from the operative field.

Any method producing an effective sympathetic block may be employed to

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produce the hypotensive state. A hyperbaric solution of procaine or tetracaine may be administered as a single injection. With the patient in the Trendelenburg position the level of anesthesia ascends. The cervical portion of the spinal canal is protected from an effective concentration of the anesthetic solution by flexion of the neck. Technics utilizing continuous spinal anesthesia with the malleable needle or the ureteral catheter, are equally effective. Epidural block, either by single injection or the continuous catheter technic, may also be used. The value of an epidural block depends upon the area over which the drug will diffuse. Injection is made at the eleventh thoracic segment. The level of anesthesia can be controlled by regulating the volume of anesthetic solution injected.

Gillies[9] states that to obtain the fullest benefits from spinal block it is necessary to insure sympathetic paralysis at a high level, rather than the level required for surgical anesthesia. For example, in pelvic surgery, if low spinal anesthesia is used, vasodilatation occurs in the anesthetized area and compensatory vasoconstriction above the level of anesthesia increases bleeding into the operative field. Sympathetic paralysis, effected by high spinal block, together with elevation of the pelvis, produces maximum benefits of the hypotensive technic.

The question arises, to what level should the blood pressure be allowed to fall? In the opinion of Gillies adequate circulation can be maintained with a systolic pressure of 60 mm. of mercury. Capillary circulation, at this level, is sufficient for cellular respiration and metabolism in vital organs provided, that (1) the blood is well oxygenated, and (2) vasodilatation is insured. Enderby[7] cautioned against an in-between range, for example, a systolic pressure of 90 mm. of mercury. At this pressure bleeding will still occur, since there is not sufficient vasomotor control to combat it. If prolonged, bleeding is more dangerous than in the individual whose vasomotor system is under normal control. The optimum systolic blood pressure is considered to be between 60 and 80 mm. of mercury.

The real hazard of the hypotensive state is anemia of the medullary centers. The Trendelenburg position will aid venous return and maintain adequate cerebral circulation. Vasopressor drugs, if indicated, should be directed toward the restoring of blood pressure to the optimum hypotensive level and not to the normal range.

From the 25 cases embodied in this report the following 2 cases are presented to illustrate the versatility of the technic:

Case 1: J. G., an 89 year old Negro man, complained of abdominal pain with nausea and vomiting for 24 hours. The patient had an appendectomy 15 years ago followed by a ventral hernia. He had a hemiplegia 15 years ago with complete recovery; he has been a known diabetic for the same period of time. On physical examination, the systolic blood pressure was 160 mm. of mercury and the diastolic pressure was 100 mm. of mercury. There were rales in both lung bases, a grade I systolic murmur and a tender mass at the site of a healed McBurney incision. The weight of the patient was estimated to be 200 pounds. Pertinent laboratory findings indicated a 4 plus albuminuria, a blood sugar of 188 mg. per cent, and a hemoglobin of 100 per cent. A flat roentgenogram of the abdomen demonstrated gas in the small bowel. A diagnosis of incarcerated hernia, with possible strangulation, was made.

Hypotensive spinal anesthesia was considered and a lumbar puncture was made at the third lumbar interspace. Through a 16 gauge Huber needle, a number 4 ureteral catheter was

inserted into the subarachnoid space. A 2.5 per cent solution of procaine in Ringer's solution was used for an anesthetic. No vasopressor drug was given. The patient was placed in the Trendelenburg position following an initial injection of 100 mg. of procaine. The blood pressure was not obtainable following this injection. The skin remained warm and dry, respirations were full and regular and the pulse rate was 80. The operation was started and the condition of the patient remained unchanged. After 35 minutes he became restless. Pentobarbital sodium, 50 mg., was given slowly intravenously. The patient then became unresponsive, but there was no change in the pulse or respiration. It was believed that because of the depressed state a vasopressor drug was indicated. Methamphetamine 4 mg. was given intravenously. The systolic pressure immediately rose to 50 mm. of mercury and the diastolic rose to 40 mm. of mercury. Twenty minutes later, additional anesthesia was required and restlessness returned. No additional sedation was given. The blood pressure remained stable at the optimum throughout the remainder of the operation.

Oxygen was administered during the postoperative recovery period. The patient was responsive 15 minutes after the end of the operation. At that time he was alert and able to sit up in bed. Mild nausea and vomiting occurred on the fifth postoperative day, associated with abdominal distention. This improved in 24 hours without recurrence. Sutures were removed on the eighth postoperative day and he was dismissed on the eleventh postoperative day.

In the presentation of this case, several facets should be discussed. No effort was made to raise the blood pressure even though it was unobtainable during the first 35 minutes of the operation. The pulse was full, strong and regular, respiration was deep and regular, the skin was warm and dry and the general condition of the patient was satisfactory. He was awake when the operation began. Oxygen was given, by mask, during the operation. Because of restlessness, a barbiturate was given intravenously, following which he could not be aroused. It was the opinion of the anesthesiologist that the circulation was too depressed and that the blood pressure should be raised to prevent the possibility of circulatory collapse.

Restlessness was noted at times throughout the operation. It was thought that restlessness may have been due to cerebral anemia or anoxia. There was no evidence during the postoperative period that any cerebral damage was sustained by the patient. The operation required 2 hours and 19 minutes and 150 mg. of procaine were used. Blood loss during operation was estimated to be 75 cc. Following the injection of methamphetamine, the blood pressure became stabilized and remained at a consistent level until the operation was completed. Forty-five minutes postoperative, the systolic pressure was 70 mm. of mercury. Three hours later the systolic pressure was 140 mm. of mercury.

Case 2: J. H., a 61 year old, obese white man, complained of weakness, nausea and vomiting, of three weeks duration, with severe, localized low back pain of seven days duration, and one episode of hematuria. The systolic pressure was 110 mm. of mercury. The pulse rate was 88. On physical examination emphysema was noted and epigastric tenderness was elicited on deep palpation. A fibrotic mass, in the left chest, with atelectasis was observed on roentgenograms and thought to be neoplastic. A diagnosis of gastric carcinoma was made. On the nineteenth hospital day, because of deepening jaundice, an exploratory laparotomy was done.

Continuous epidural anesthesia, with 1 per cent solution of lidocaine, was used. No vasopressor drug was given. Fifteen minutes was required for the onset of anesthesia following an initial injection of 30 cc. of the solution. The systolic pressure fell from 100 mm. of mercury to 70 mm. of mercury and the pulse rate was 80. Oxygen was administered, by mask, throughout the operation. The systolic pressure fluctuated at first, but later stabilized at 60 mm. of mercury. Restlessness was controlled with 10 mg. of Benedryl intravenously. The operation lasted 2 hours and 55 minutes. The estimated blood loss was 55 cc. After a stormy postoperative course, the patient died on the twenty-eighth postoperative day of generalized carcinomatosis.

These 2 cases demonstrate that hypotensive anesthesia can be tolerated by poor-risk patients having major operations. Blood loss was minimal in each case and postoperative complications, relative to anesthesia, did not occur. The patients remained alert and oriented except when depressed with drugs. No cyanosis was observed and the skin remained warm and dry. In each patient, the blood pressure slowly returned to the normal preoperative level. Nausea has occurred in several patients following induction of the hypotensive state and during operation when vagal reflexes were elicited by traction.

Postoperative hemorrhage occurred in 1 patient who had had a combined abdomino-perineal resection and developed shock. Shock was controlled with 1 pint of blood and recovery was without further complication. It is of interest to note that when shock became apparent in this case it was accompanied by a rise in blood pressure and not a fall. Clinically, there was adequate kidney function and all the patients voided spontaneously immediately following operation.

DISCUSSION

Several theories have been postulated to explain the mechanism whereby the hypotensive state is produced. The following are the most plausible.

Co Tui[2] has reported that following paralysis of the vasoconstrictor nerves, the systolic blood pressure rarely fell below 50 mm. of mercury unless there was an accumulation of carbon dioxide superimposed on the vasoconstrictor paralysis. This was said to cause the loss of muscular tone in the vessel walls resulting in an additional pressure drop which might reach zero. Carbon dioxide lowers the blood pressure by its toxic action on the sympathetic nervous system which is particularly vulnerable during spinal anesthesia. This results in peripheral capillary dilatation and a fall in blood pressure.

Co Tui[3] divides the course of circulatory events following the injection of an anesthetic agent into the subarachnoid space in dogs into three phases.

Phase I. A primary fall occurs which is accompanied by a rise in the volume and temperature of the lower extremities. This is attributed to paralysis of the vasoconstrictor nerves in the regions affected immediately. The vessels become relaxed so that the volume of circulating blood at that moment is inadequate for the enlarged vascular tree.

Phase II. Following Phase I, the carotid sinus reflex is called into play. The heart rate is increased and the unaffected vessels constrict to reduce the capacity of the vascular tree, insofar as this is possible, and a compensatory rise in blood pressure occurs. As the drug spreads and the number of nerves becoming paralyzed increases, the blood pressure gradually falls again.

Phase III. As a gradual and prolonged fall in pressure occurs, the intercostal muscles become paralyzed which leads to anoxia and the accumulation of carbon dioxide. This further adds to the fall in pressure.

There is still much controversy concerning the actual mechanisms involved because no direct correlation can be made between the results obtained from the animal experiments and the physiologic changes brought about in human subjects.

As early as 1900, Tuffier[13] observed a fall in blood pressure during spinal anesthesia. He attributed the hypotension to paralysis of the splanchnic nerves with a pooling of blood in this area. In 1915, Porter and Smith[11] attributed the hypotensive state to intercostal paralysis. In 1932, Bower[1] and coworkers suggested that the fall in pressure was due to paralysis of the cardiac nerves. Smith and coworkers[12] in 1939, reported work to substantiate the theory that the blood pressure fall in spinal anesthesia results *not* from arteriolar dilatation, but from a reduction in cardiac output in consequence of venous stagnation. It was explained that when a fall in blood pressure occurs it is the result of a decrease in the cardiac output with an unchanged arteriolar bed. The cardiac output is actually decreased, due to a decreased stroke volume resulting from postarteriolar dilatation and decreased venous pressure. This can be explained in terms of dilatation of capillaries, venules, and veins following skeletal muscle paralysis, without definitely proving the existence of tonic sympathetic activity in these circulatory channels.

It is not always necessary or desirable, to superimpose general anesthesia upon the hypotensive patient. He experiences no subjective sense of change in blood pressure and is alert and rational throughout the operation. This is helpful to the anesthesiologist for the sensorium may serve as a valuable index to the patient's general welfare. If syncope occurs, adjustment of the table to improve venous return will alleviate this symptom. Nausea may occur during abrupt changes in blood pressure. This serves to alert the anesthesiologist to the possibility of vomiting, which if it occurs, is not as dangerous to the patient as it might be if he were rendered unconscious by general anesthesia. Restlessness may be the result of hypoxia or the buildup of carbon dioxide if respiratory exchange is inadequate. Oxygen is, thereby, indicated and should be administered. If the patient becomes disoriented or fails to respond, the anesthesiologist can detect the change in the sensorium and look for the cause. Masking a high spinal with general anesthesia removes the most valuable signs in the clinical evaluation of the patient under spinal anesthesia.

During the hypotensive state the patient is calm and quiet. The skin remains warm and dry and the peripheral veins are well filled. The skin although pale, is of good color. In essence, then, the appearance of the hypotensive patient is not unlike that of the normotensive state.

Should the need for depressants arise during hypotensive anesthesia, the usual dosage may or may not be required. It is believed that during the hypotensive state the metabolic rate is lowered, a condition which of itself has a depressant effect; therefore, opiates and barbiturates must be given slowly and in divided doses, with careful observation of the pulse, blood pressure and the sensorium.

If a vasopressor drug is used at any time during the procedure, slow intravenous injection of a dilute solution is recommended to avoid a sudden rise in the blood pressure. Delicate thrombi that may have formed in vessels at the operative site may be dislodged by a sudden increase in systemic pressure, giving rise to severe bleeding. This point may be overlooked by the surgeon who wants the blood pressure back to normal before closing the wound, defeating the purpose for which

the hypotensive technic was designed. The blood pressure rises slowly during the postoperative period provided there has been little blood loss, and adequate blood replacement has been made.

It is emphasized that the primary objective of the hypotensive technic is directed toward the conservation of blood. Theoretically, if hypotensive anesthesia is carefully controlled, blood loss will be insignificant. This is only true in the patient coming to surgery with a normal blood volume. Preoperative transfusions are strongly recommended for patients who have lost blood or who are anemic. A careful estimate of blood loss during operation is necessary in order that adequate replacement may be made, because the safe and efficient use of the hypotensive technic is based upon a normal circulating blood volume.

Complications arising from the use of this technic fall into the same category as those for spinal anesthesia. Additional hazards result more often from inattention to the vital signs rather than from the technic itself. Shortness of breath may become a complaint if all of the intercostal muscles are paralyzed. This is aggravated by the weight of the viscera against the diaphragm while the patient is in the Trendelenburg position. Adjusting the table to a more horizontal position may be sufficient to make the patient more comfortable. Even if the level of anesthesia should rise high enough to paralyze the vocal cords, this does not constitute an insurmountable hazard, per se, if efficient means of ventilating the patient are immediately available.

The appearance of the patient during the hypotensive state is such that the uninformed observer would not suspect a low blood pressure. The fact that the patient himself is not aware of the change tends to indicate that no great physiologic disturbance has taken place. It is true that the flow of urine often ceases while the blood pressure is low, but it is the belief of those who are familiar with the method that the kidneys are not deprived of an adequate circulation nor does tissue anoxia occur. Postoperative anuria has not been observed in any of these patients, although a normal output may not be established for 24 hours. As is true with spinal anesthesia in general, morbidity due to the anesthesia is low. Patients tend to recover quickly and easily. Poor-risk patients often make better progress because of improved circulation and adequate oxygenation. It has been shown in animals that the work load on the myocardium is reduced 56 per cent, whereas the coronary flow is decreased by only 25 per cent; thus the coronary flow becomes more abundant relative to the cardiac needs.[5]

One patient having congestive failure with ankle edema and ascites came to the operating room in a semi-sitting position because he was unable to breathe comfortably in the supine position. The blood pressure fell following spinal anesthesia, the pulse slowed, the patient became more comfortable and was able to rest quietly on his back in a moderate Trendelenburg position. As the effect of the anesthesia subsided, he requested to be placed in a semi-sitting position to facilitate respiration. This is only *one* case, but it illustrates that a patient in congestive failure may tolerate the hypotensive state comfortably without apparent untoward effect. Clinically, it would seem that the condition of such a

patient is actually improved during the hypotensive state. If this be true, such a method may prove to be valuable in selected cases.

From this discussion it is *not* to be inferred that the hypotensive technic is without danger. Dripps and Vandam[4] stated that "in patients with low blood pressures, lesions of the brain, kidneys, heart and liver have occurred as the result of either an inadequate flow of blood through the patient's vessels or occlusion of vessels by spasm or thrombosis. If blood vessels are sclerotic or otherwise abnormal, calibre changes in response to the hypotensive state may not occur as readily as in normal vessels. If this occurs, circulation through these vessels would be impaired." However, much of the criticism based on theoretical grounds does not necessarily hold. Neither does this small series of cases disprove the presence of overt danger. It is believed, however, that, since poor-risk patients can be successfully managed by employing the hypotensive technic, this method may have some merit when applied to selected cases.

CONCLUSIONS

We have demonstrated that a few aged, poor-risk patients did tolerate the hypotension remarkably well, and did not suffer any untoward effects that could be related to the type of anesthesia or that could be detected clinically. On the contrary, these patients all recovered quickly and all but 1 made excellent postoperative progress.

Again, we emphasize that no attempt is made to draw conclusions from these few observations. We report our observations literally from the bed-side, to gain what knowledge is to be had from the clinical experience.

What criteria then serve for the selection of patients for hypotensive anesthesia? Any patient for whom large blood loss may be anticipated is a potential candidate for the method. Also suitable is the patient scheduled for lower abdominal surgery when the Trendelenburg position is of advantage to the surgeon.

Less suitable is the patient who must remain in a horizontal position or when positioning places the center of gravity below heart level. Venous return is dependent upon gravity and it would seem unwise to subject a patient to a state of hypotension unless the Trendelenburg position is used.

The anemic, debilitated patient with reduced blood volume is a poor candidate for the hypotensive state. For this type of patient adequate preoperative transfusion is deemed advisable and is strongly recommended.

This paper has been presented primarily to show that the hypotensive technic may be carried out on the elderly and the poor-risk patient. Whether or not these patients might have done better under some other form of anesthesia is a matter of conjecture. No complications were directly attributable to the anesthetic. Since this series is small, the method cannot be recommended on a statistical basis.

Whether or not the kidneys are harmed by a marked fall in blood pressure is still under investigation.

It would seem that patients with hypertensive cardiovascular disease tolerate

the hypotensive state well. As for the sensorium, all patients were alert and oriented during the postoperative period.

The hypotensive state may be tolerated reasonably well in some patients, but further evidence is sought to obtain a more critical evaluation of this method of anesthesia.

Regardless of favorable opinions which may be presented in defense of the hypotensive technic, its value has not been proved beyond a doubt. Unseen dangers may lurk in the shadows.

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DIVERTICULOSIS AND DIVERTICULITIS OF THE VERMIFORM APPENDIX

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Diverticulosis and diverticulitis of the vermiform appendix are among the less common conditions involving this vestigeal organ. Prior to 1900 the occasional references to the subject were made by the European pathologists; however, as the true nature of acute appendicitis became more widely recognized and surgical removal of the appendix became more common, the subject of appendiceal diverticula became of more interest to both surgeon and pathologist. One of the earliest descriptions of appendiceal diverticula appeared in Edel's[4] paper of 1894. In 1923 Stout[18] made a thorough study of the subject, pointing out that the condition occurred more frequently than had previously been thought and reported some experimental work endeavoring to show how diverticula are formed. For a review of the historical background of this subject the reader is referred to the excellent paper by Collins.[2]

It would seem that such a condition as appendiceal diverticulosis, being anatomically obvious and etiologically uncomplicated, would be thoroughly understood. However, such is not the case, for there is considerable discrepancy in the reported incidence as well as controversy over classification and pathogenesis. Having recently operated upon a patient with the preoperative diagnosis of acute appendicitis only to find a noninflamed appendix with multiple diverticula, one of which was acutely inflamed and perforated, we have reviewed the available literature on the subject and studied the records of the University of Kansas Medical Center for similar cases.

INCIDENCE

Although not entirely a rarity, diverticulosis of the appendix is uncommon enough to excite considerable interest when encountered at operation. Collins, in reviewing the literature, found 67 cases in 16,044 appendices studied (both surgical and postmortem specimens), or an incidence of 0.42 per cent. To this series he added 30 cases of his own occurring in 4,071 appendices studied, or 0.74 per cent. Stout was one of the first to point out that these lesions are frequently missed by the surgeon as well as by the pathologist unless careful search is made. Ladin,[13] in a recent review, reports 16 cases, only 4 of which had been noted by the surgeon. He also reports that only 42 cases were found in 51,525 appendices studied at the Army Institute of Pathology, an incidence of 0.08 per cent. At the University of Kansas Medical Center only 2 cases of appendiceal diverticula have been reported in approximately 3500 patients whose discharge diagnoses are recorded under appendix or appendiceal disease. It would then

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appear that the true incidence is approximately 0.5 per cent, or 1 case of diverticulosis in every 200 appendices.

PATHOGENESIS AND CLASSIFICATION

There is considerable discussion in the literature concerning the exact manner in which appendiceal diverticula are formed, and the few attempts to form diverticula experimentally have thrown scant light on the subject. Consequently any classification of these lesions is open to serious question. It would seem however, that the following classification is as satisfactory as any:

I. Congenital
II. Acquired
  a. Inflammatory
  b. Noninflammatory

Congenital diverticula of the appendix are those in which all layers of the bowel wall are retained intact in the diverticulum, while in the acquired type there is no muscularis layer, the walls of the diverticulum consisting only of mucosa, submucosa and serosa. Those of congenital origin are rare and several cases reported in the literature as such, are open to question.[1, 6, 12, 14] The case reported by Hedinger,[10] found at autopsy in a stillborn infant as well as 2 cases reported by Sturm[19] under similar circumstances, are without question truly congenital. One of the 25 cases reported by Wilson[20] is of congenital origin.

The acquired type of diverticula may be inflammatory or noninflammatory in origin, although it is often difficult to determine the precise manner of formation in any given case. Stout was one of the first to investigate these lesions, doing thorough gross and histologic studies of 7 cases and later attempting to reproduce diverticula in experimental animals. He concluded that in order for a diverticulum to form there must be a weak point in the muscular wall of the appendix either due to a defect where the blood vessels enter or a scarring and thinning of the muscularis resulting from a suppurative process or previous rupture. Edwards,[5] who made an exhaustive study of diverticulosis and diverticulitis of the intestinal tract, reached much the same conclusion as did Stout. However, he also postulated a prolonged contraction of the longitudinal muscle layer causing a shortening of the tube and consequent foldings or sacculations of the mucosa which were then caused to herniate through the muscle by contractions of the circular coat. Kline, Young and Straus[11] studied the largest series of cases reported and concluded that most of their cases were the result of extension of an inflammatory process through the appendiceal wall causing necrosis, liquifaction and cavitation of the adipose tissue. As the process heals the mucosa, submucosa and serosa grows into the cavity with a resultant diverticulum having no muscularis. Similar conclusions were reached by Gramse, Dockerty and Waugh.[9] On the other hand Wilson presents a long and thorough discussion of the subject including attempts to reproduce diverticula in recently removed human appendices and concludes that inflammation plays little or no part in the development of the common types of appendiceal diverticula except

for the production of a constriction of the appendiceal lumen. It is his opinion that stenosis of the lumen leads to distention of the appendix and that subsequent muscular contractions result in a herniation of the mucosa. Certain authors[3, 5, 20] have speculated on the possible significance of an increased number of neuromuscular complexes often observed in fibrotic appendices, but there seems to be little reason to associate these findings with the development of appendiceal diverticula.

CLINICAL FEATURES

The clinical diagnosis of appendiceal diverticulosis or diverticulitis is seldom made. In fact the presence of small diverticula is often overlooked by the surgeon at operation and most of the reported cases have been found by the pathologist. Stout, Ladin and others have pointed out that careful search by the pathologist undoubtedly would reveal a higher incidence than presently reported. Gilmore and Mahon[8] report 1 case demonstrated roentgenographically. There are no clinical findings which might help in making the diagnosis preoperatively, although Sauer[17] makes a point of the slow and gradual onset of symptoms in the right lower quadrant. Edwards has pointed out that the occurrence of appendiceal diverticula is not merely an aspect of diverticulosis of the colon, since most of the cases in which the appendix is involved show no evidence of involvement of the large bowel. He also found that the average age of patients showing appendiceal diverticula is considerably less than those of colonic diverticulosis. Collins found the average age of 54 patients with appendiceal diverticula to be 35.7 years, while in Edwards' series the average age was 42.

It long has been known that appendicitis is more apt to occur in a patient having had a previous perforation treated by simple drainage of an abscess or by conservative nonoperative management; likewise, such cases usually are more fulminant in nature than those having no such previous history. This increased liability to subsequent attacks in a patient who has had a previous perforation has been explained by Pack[16] and others[2, 9] as being due to diverticulum formation following the previous perforation. Likewise the incidence of perforation and peritonitis in such cases is higher because of the thinned out diverticular wall. Gardham, Choyce and Randall[7] report the occurrence of pseudomyxoma peritonei following rupture of an appendiceal diverticulum and speculate at length on the connection between the two conditions.

CASE REPORT

L. J. H., a 57 year old Negro man entered the Kansas City Veterans Hospital on Jan. 8, 1953 complaining of abdominal pain of four days' duration. The symptoms had started as generalized abdominal discomfort accompanied by nausea and vomiting. He took castor oil but did not improve and the abdominal symptoms became localized in the right lower quadrant. The past history was significant in that the patient had been hospitalized at this hospital two months previously because of hemorrhoids. At that time a barium enema was given and no abnormalities were found in the roentgenogram. There was a known history of syphilis with minor tabetic changes. There was no history to suggest previous attacks of appendicitis.

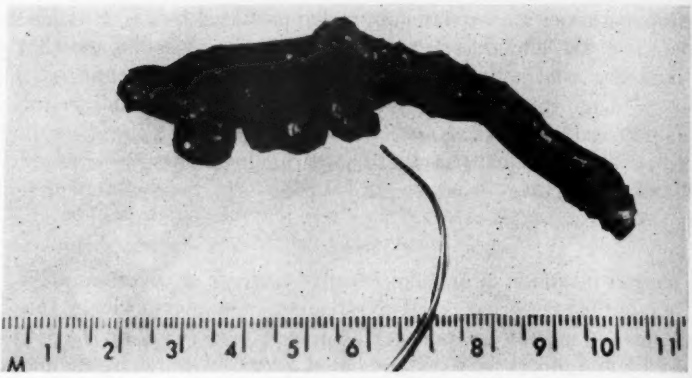

FIG. 1. Photograph of appendix showing multiple diverticula, the most proximal of which has ruptured.

Physical examination: Pertinent physical findings were confined to the abdomen which showed marked right lower quadrant tenderness, right rectus spasm, rebound tenderness and referred tenderness to the right lower quadrant. There was no tenderness on rectal examination. The temperature was 98.6 F.

Laboratory Findings: The white blood count was 20,100 per cu. mm. with 90 per cent neutrophils; hemoglobin 14.5 Gm. per 100 cc. Urinalysis showed 1 plus albumin and 80 to 100 white blood cells per high power field with some clumping.

Preoperative Diagnosis: Acute appendicitis.

Operative Findings: Under spinal anesthesia a right lower quadrant, muscle splitting incision was made. There was a small quantity of purulent exudate present on opening the peritoneum. A culture was taken. The appendix was found to be in a retrocecal position. When this was exposed by elevating the cecum into the incision, there were found to be five diverticula projecting from the anti-mesenteric border. The most proximal of these was acutely inflamed and showed a perforation 4 mm. in diameter. The appendix was removed and the abdomen was closed without drainage.

The postoperative course was uneventful, and the patient was discharged on the tenth postoperative day. The smear and culture taken at operation showed gram negative rods of the coliform group. A repeat urinalysis was normal. A follow-up barium enema again was normal with no evidence of diverticula of the colon. Barium studies of the stomach and small bowel also were reported as showing no abnormalities.

Pathologic Study: The removed specimen showed an appendix measuring 8 cm. in length with five diverticula projecting from the anti-mesenteric border along the distal half of the appendix (fig. 1). The largest of these was 9 mm. in diameter and all communicated with the appendiceal lumen which was patent throughout. The most proximal diverticulum was acutely inflamed and showed a perforation 4 mm. in diameter at its end from which purulent material exuded on pressure. Microscopic studies revealed a normal appendiceal mucosa with no suggestion of acute or chronic inflammation. The perforated diverticulum showed acute hemorrhagic inflammatory changes and the other diverticula showed extensive scarring and round cell infiltration. The diagnosis was diverticulosis of the appendix and acute diverticulitis with perforation and peritonitis.

SUMMARY

Diverticulosis and diverticulitis of the vermiform appendix while not rare, are uncommon enough to excite considerable interest when encountered at operation.

The true incidence is approximately 0.5 per cent although many cases are not recognized by the surgeon and some are overlooked by the pathologist.

The exact nature of these outpouchings remains controversial, although it is clear that appendiceal diverticula are not merely an extension of diverticulosis of the colon.

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ENTERIC INTUSSUSCEPTION

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Enteric intussusception is an invagination or telescoping of the small intestine into itself. It has been further subdivided into jejuno-jejunal, jejuno-ileal, ileo-jejunal (retrograde) and ileo-ileal. As there is no signpost dividing jejunum and ileum, it seems preferable to class all small bowel intussusceptions under the term *enteric* and localize the distance of it from the ileocecal valve or the ligament of Trietz.

Intussusception has been considered a surgical curiosity in the adult.[4, 8, 9, 15, 19] Most pure enteric intussusceptions occur in older children and adults, and are usually due to intraluminal tumors.[4, 9, 21]

A personal series of cases of enteric intussusception are herein presented. It is an uncommon form of intestinal obstruction, the signs are confusing and the diagnosis is difficult.[6, 16, 23] This presentation attempts to focus attention on whatever these cases have in common to help determine a means by which earlier diagnosis may be established and rational therapy instituted.

CASE REPORTS

Case 1. Enteric intussusception with two benign polyps causing abdominal mass. G. W. W., a 5 year old white boy was admitted to the Pineville Charity Hospital on Sept. 18, 1946 with intermittent abdominal cramps, vomiting, diarrhea and "knots rolling around" in his abdomen for two weeks duration. Two years previously he had passed a long worm. The patient doubled up and rolled around in bed and occasionally would lie face down, knees drawn under his abdomen. The abdomen was flat and soft. A mass measuring 5 cm. by 3 cm. was palpated to the left of the umbilicus. The mass was not tender and it seemed firmer in some areas than in others. Roentgenograms of the abdomen with the patient flat and erect were negative and a barium enema did not show pathology of the colon or rectum.

Operation: On September 23, the abdomen was explored through a left paramedian incision. A large mass of intussuscepted small intestine was found beginning 20 cm. distal to the ligament of Trietz. The intussusception was reduced by taxis and it proved to be compound. A tumor was palpated after reduction. The intestine was opened and two mucosal polyps were found (fig. 1.). Resection and end to end anastomosis of the polyp-bearing area was done after complete exploration revealed no other pathology. The patient recovered rapidly and was discharged on the tenth day.

Case 2. Jejunal leiomyoma causing chronic and recurrent enteric intussusception in an adult. A. de M., a 57 year old white woman was admitted to the Lakeshore Hospital, New Orleans, Louisiana on Dec. 23, 1948 with epigastric pain and history of vomiting for 24 hours. Indigestion, gas, bloating, epigastric pain and abdominal cramps had been troubling her for the past three months. A gastrointestinal roentgenographic survey two months before hospitalization was negative except for spastic colon and evidence of appendicitis.

An appendectomy had been done on Nov. 12, 1948 and the specimen was reported "chronic, fibrous; evidence of an old healed subacute episode." Abdominal examination showed moderate distension and increased peristalsis. Abdominal roentgenograms taken

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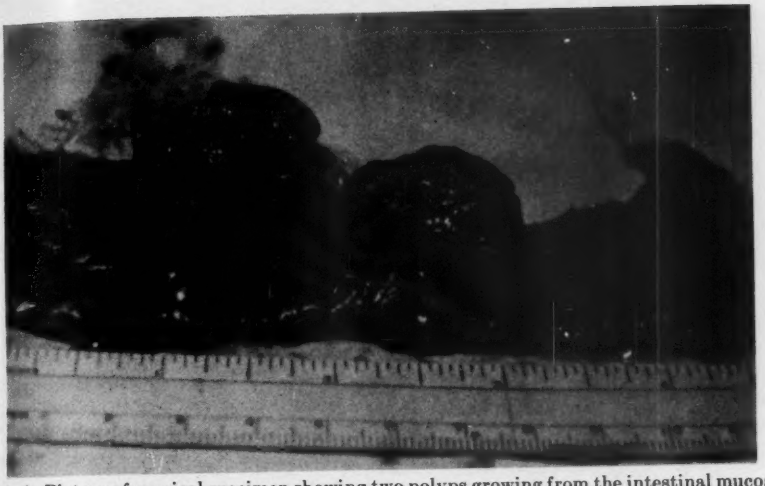

FIG. 1. Picture of surgical specimen showing two polyps growing from the intestinal mucosa

FIG. 2. Jejunal polyp removed by author in case 2

in erect and flat postures showed no evidence of intestinal obstruction. Wangensteen suction and intravenous fluids provided immediate relief after 12 hours. A gastrointestinal roentgenographic series with orally administered barium sulfate on December 24 and December 30 showed "a smooth regular dilated segment of bowel near the upper jejunal loop with proximal and distal areas of constriction, indicating an obstructing lesion in the upper jejunal loop."

Operation: On Jan. 5, 1949, the abdomen was opened through a left rectus incision and a jejunal intussusception, 15 cm. distal to the ligament of Trietz, was found and reduced by taxis. A mass was palpated in the intestine at the leading point. The intestine was opened and a jejunal polyp was found (fig. 2). Excision and primary end to end anastomosis was done after further exploration proved negative. The patient recovered and was discharged on the ninth day. She has remained free of any gastrointestinal disturbances.

Case 3. Compound enteric intussusception of jejunal carcinoma, massive resection of small intestine and mesentery containing metastatic lymph nodes: a five year survival. J. C., a 24 year old white woman was hospitalized in the Hotel Dieu, New Orleans, Louisiana on Nov. 22, 1948 with recurrent attacks of colicy abdominal pain radiating to her back. She was vomiting thick green fluid and had been sick for the past 24 hours. Similar attacks of colic had occurred in the past two years, becoming worse during the last six months, during which time she lost 25 lbs. A uterine suspension and an appendectomy had been done two weeks prior to admission with no relief.

Laboratory data showed anemia, hypochloremia, and hypoproteinemia. The serum chlorides were 363 mg. and the total proteins 5.3 Gm.

Roentgenograms of the abdomen and retrograde pyelograms were negative. Gastrointestinal roentgenograms with orally administered barium sulfate revealed "impediment of the flow six inches distal to the ligament of Trietz and a filling defect in the lumen about 18 inches from the ligament." Dehydration, electrolyte imbalance and anemia were corrected with intravenous fluids, electrolytes, and blood transfusions.

Operation: On December 20, the patient's abdomen was explored through a left rectus incision and a compound intussusception involving the jejunum and part of the ileum was found (fig. 3). Reduction was accomplished by taxis and there was a firm, umbilicated tumor in the wall of the jejunum acting as the leading point (fig. 4). Frozen section biopsy of an adjacent mesenteric lymph node was reported to contain metastatic carcinoma. Lymph nodes proximally and distally were studied until a negative report was returned. This point was used to determine the extent of small bowel resection. Massive resection of the small bowel and lymph node-bearing mesentery was done. An end to end anastomosis of the remaining 4 inches of jejunum and 12 inches of terminal ileum was made. Pathology report: "Adenocarcinoma of the jejunum, grade III, with invasion to the underlying serosa, and massive metastasis in the mesenteric lymph nodes. A lymph node from the mesentery of the ileum shows no metastasis."

Postoperative diarrhea was controlled with paregoric and amphojel. On the twelfth postoperative day, paregoric was discontinued because of constipation requiring enemas. She was discharged on Jan. 19, 1949, having two soft stools daily, and having gained 14 lbs. from a weight of 84 lbs. before operation. She continued to gain weight at home and a follow-up roentgenographic gastrointestinal series five months after operation was reported as follows: "There has been an anastomosis done between the proximal jejunum and the distal portion of the ileum. The intervening intestine has been removed leaving approximately 3½ inches of jejunum and about 14 inches of terminal ileum. At one hour after the barium meal the stomach is practically empty and a portion of the barium meal has reached the rectum. At two hours there is only a small quantity of barium left in the small intestine, the remainder being distributed in the colon. At six hours there is only a minimal quantity of barium in the small bowel—at the site of the anastamosis."

After five years there is no evidence of recurrence, and an average weight of 115 lbs. is easily maintained. The patient can eat any food without discomfort or diarrhea.

Case 4. Idiopathic enteric intussusception during pregnancy. D. Z., a 19 year old white married prima para of five months was admitted to the Montelepre Memorial

FIG. 3. Compound intussusception of jejunum and ileum found in case 3

FIG. 4. Umbilicated tumor in wall in jejunum causing intussusception in case 3. (Adenocarcinoma of jejunum, grade III, massive metastases to mesenteric lymph nodes.)

Hospital, New Orleans, La. on June 24, 1953, with right umbilical pain radiating down the lower abdomen toward her right leg. For the past 12 hours she had experienced intermittent cramping, nausea and vomiting. She had had similar attacks for the past six years with pain that "doubled her up." A diagnosis of appendicitis had been made during one such episode and appendectomy advised. The abdomen was flat, with right sided pain, tenderness, some muscle guarding and moderate localized rebound tenderness. The uterine fundus was palpable just above the umbilicus. Laboratory studies showed polymorphonuclear leukocytosis and a mild anemia. Urinalysis was negative for sugar albumin and pus cells. A diagnosis of "Appendicitis, complicating pregnancy" was made.

First operation: An appendectomy was done through a right rectus incision. The appendix was not acutely inflamed so the terminal ileum was explored and no Meckel's

diverticulum was found. Her symptoms were ascribed to pregnancy and the abdomen was closed. The first two days were uneventful and a soft diet was retained. On the third day there was nausea, generalized abdominal pain and restlessness controlled with sedation. Distention became apparent on the fourth day. She vomited bile and hyperperistalsis was auscultated alternating with long silent intervals. Relief was obtained with Wangensteen suction, intravenous fluids and electrolyte solutions. On the sixth day, the abdomen again became distended, she vomited continuously and much hyperperistalsis was heard. Abdominal roentgenograms in erect and flat postures showed distended loops of small intestine with fluid levels. Mechanical intestinal obstruction was diagnosed and fluid, electrolytes and blood were given intravenously.

Second operation (idiopathic intussusception): The patient's abdomen was explored by reopening the original right rectus incision. Loops of distended small intestine presented. A loop of collapsed intestine was found and traced proximally to the point of obstruction —an enteric intussusception about 90 cm. past the ligament of Trietz. The intussusception was easily reduced and careful palpation revealed no underlying pathology as the cause, so the abdomen was closed.

The patient improved rapidly during the first day. However, distension remained unrelieved during the second day in spite of continuous Wangensteen suction. Increased bowel sounds were heard and roentgenograms again showed distended small bowel and fluid levels. Reoperation was decided upon.

Third operation (ileostomy): The wound was reopened for the second time and the small intestine was found to be distended throughout. A few plastic adhesions between distended loops were noted, but were not causing any obstruction. A terminal ileostomy was made, allowing the catheter to drain through the lower end of the wound which was then closed with through and through figure of eight sutures. Abdominal distension receded rapidly and gastric suction was discontinued after 72 hours when normal peristalsis returned. The first bowel movement occurred on the fourth day, the catheter was withdrawn on the eighth day and the patient was discharged on the ninth postoperative day. She has had no further difficulties, and delivered a boy normally on Oct. 25, 1953.

DISCUSSION

These cases histories bring up certain points of diagnosis and therapy that are worth mentioning.

Difficulty in diagnosis was the general rule in each case. Each patient, except the one in case 1, received some form of surgical treatment other than that finally justified. Case 2 first had an appendectomy; case 3, a uterine suspension and appendectomy, and case 4, an appendectomy.

A tumor mass was present on physical examination only in case 1. The presence of this palpable abdominal tumor influenced prompter surgical intervention than in the other 3 patients.[13]

Roentgenologic examination was found to be a valuable adjunct in diagnosis.[1, 2, 4] However, certain limitations should be mentioned:

1. Abdominal films with the patient in erect and flat postures were of no value in the first 3 cases because of the incompleteness of the obstruction. Distended small bowel pattern and fluid levels were of much value in deciding to re-explore in case 4.
2. A barium enema roentgenogram is conclusive evidence in the type of intussusception which involves the large intestine. A characteristic cupola type of filling defect is shown. However, a negative roentgenogram of a barium enema does not rule out the presence of the enteric variety of intussusception. This was demonstrated in case 1.

3. Orally administered barium was found to be of value in localizing the lesion in cases 2 and 3. It should be mentioned that it is not without danger to administer barium orally in the presence of intestinal obstruction, because of possible rupture of the intestine.

Intraluminal tumors were the underlying cause of intussusception in 3 of the 4 cases reported. This follows the general rule that in almost all cases of intussusception in older children and adults there is some intestinal lesion which produces the intussusception by acting as the leading point.[3, 4, 8, 9, 10, 13, 14, 15, 16, 17, 19, 20] Intestinal polyps, Meckel's or other diverticula, bowel duplications, lipomas, carcinomas, lymphosarcomas, and other benign or malignant neoplasms are the most common causative lesions. It has been a surgical axiom to meticulously search for an underlying cause whenever intussusception occurs in the adult.

Idiopathic intussusception (case 4) is generally considered a surgical curiosity in the adult. Baily[3] mentions that Mooro recorded 2 cases which occurred in Cairo during the Mohammedan fasting season and presumed the abnormal peristalsis caused by the pangs of hunger determined the invagination.

Intussusception during pregnancy (case 4) is considered a rarity. Chaffin, Mason and Slemons[5] reported 1 case of intussusception during pregnancy and have collected 11 cases from the literature since 1870. The classical manifestations of parturition baffle differential diagnosis.

Ileostomy decompression[24] and drainage of the small intestine proved most practical in case 4. The five month gravid uterus seems to have acted as an external cause of obstruction after the intussusception was reduced. Pressure from the enlarged uterus delayed the return of tone to the distended small bowel. Terminal ileostomy maintained decompression until bowel tone returned. It seems worthwhile to mention the successful use of ileostomy in a case of intussusception during pregnancy as both the maternal and fetal mortalities that have been reported are well above 50 per cent.[5] Additional responsibility is placed upon the surgeon operating upon a pregnant woman, as there are two lives at stake. Chaffin and associates found it necessary to sacrifice the fetus and uterus (five months gravid) to facilitate exposure. In case 4 we were able to spare the uterus and fetus and a healthy boy was normally delivered four months after surgery. This experience indicates that the use of ileostomy decompression would allow the pregnancy to be "left alone."

Carcinoma of the jejunum (case 3) is an infrequent form of malignancy. According to Mayo and Nettrour[16] it comprises 0.15 per cent of carcinomas of the gastrointestinal tract. The case reported here came to surgery because the tumor produced an enteric intussusception which caused the symptoms of incomplete obstruction. Frozen section biopsy was utilized during the surgical procedure to determine the amount of involvement and therefore the area that required resection. Case 3 represents another five year survival after resection for carcinoma of the jejunum. This is important because it has been said that the prospect of a five year survival following resection of the small intestine for jejunal carcinoma is no higher than 5 per cent.

Massive small bowel resection has been done on occasion for regional ileitis,

obstruction with gangrene, superior mesenteric artery occlusion, volvulus, internal hernia, malignant involvement and polypoid tumors.[1, 7, 11, 12, 22] Case 3 represents a massive small bowel resection in which the patient did not develop a sprue-like syndrome or jejunal-ileac insufficiency, but was able to maintain weight, nutrition and bowel control.

CONCLUSIONS

One might justifiably conclude from the experiences herein reported that any patient suffering with recurrent bouts of colicky abdominal pain, even with negative physical and roentgenologic findings, should be subjected to a thorough abdominal exploration.

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PERINEAL HERNIA FOLLOWING ABDOMINOPERINEAL RESECTION*

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There are many books on proctology, and many articles about abdominoperineal resection, but only a very few of these mention perineal hernia as one of the postoperative complications. The first case was reported by Yoemans[11] in 1937 at the meeting of the American Proctologic Society. His case occurred four months after perineal excision of the rectum for cancer and he remarked that it was the only one he had seen in many similar procedures. The hernia was repaired from above by obliterating the sac and using the uterus to help close the ring. It had not recurred at the time of publication of his report nine months postoperatively. This case provoked much discussion at the meeting and Hayden[5, 7] mentioned having seen only 2 cases; T. E. Jones[8] had seen only 1 in 300 cases. McKenney[10] mentioned repair by ligation of the sac from below but had a prompt recurrence. Frank and Colp,[3] in 1941, reported a case following perineal excision for cancer of the rectum. This one was repaired from below but since the sac was not excised in the first procedure a second operation, also from below, was necessary to effect a cure.

Hayden, in 1939, listed perineal hernia as a complication of abdominoperineal resection. He reported that only 4 cases could be found at Massachusetts General Hospital up to that time. No operation was done in any of these because it was believed futile to attempt a construction of a new pelvic floor. These patients were very uncomfortable and did not respond to external supports of various types.

In 1944, Cattell[2] reported 1 case in 800 procedures and repaired it successfully from below by ligation of the sac, suturing the uterus to the sacrum and closing the pelvic floor beneath this.

In 1946, Jenkins[6] reported 1 case not treated. An interesting aspect of this case was a normal cystogram on both lateral and anteroposterior views. The patient had no complaints.

Bacon,[1] in 1949, added 2 cases, 1 repaired from below and 1 repaired intra-abdominally. Gabriel[4] reported 6 cases and believed that they should all be repaired from below by a technic to be discussed later.

ETIOLOGY

Because of the rarity of this condition and the infrequency of cases reported in detail, the exact cause of perineal hernia is not known. According to Cattell, removal of the coccyx definitely contributes, in some part, to the development of perineal hernia. He states that it has been their custom to close the perineal

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wound about a small cigarette drain, the upper portion of which is placed at the level of the cervix or seminal vesicles. With the coccyx present, the cervix and vagina, or vesicles and prostate gland fall back in contact with the coccyx and sacrum as soon as the drain is removed. He has seen these structures closing the pelvic outlet on re-exploration for pelvic abscess postoperatively or on re-exploration for recurrence of the malignancy. Bacon reviewed 16 cases including 2 of his own and studied them with particular reference to etiology. He concluded that wound closure or coccyx removal made no difference in the occurrence of perineal hernia.

Although removal of the coccyx may play some part in the occurrence of these hernias, it certainly is not a major factor. According to Krasky and Lockhart-Mummery[9] posterior resections were popular in the early nineteen hundreds and if perineal hernia had been frequent we believe that it would have been reported at that time. There is no record of any extensive occurrence of hernias of this type during the era when many posterior resections were done. It is evident that a combination of factors exists and is responsible for the occurrence of this type of hernia. The same factors which cause wound eventration or evisceration naturally exist since a peritoneal surface is all that prevents the abdominal contents from going to the perineal skin or into the perineal wound immediately postoperatively. These factors are severe coughing due to pneumonia, atelectasis, or cardiac conditions; any type of perineal infection or intra-abdominal infection; poor wound healing; low serum protein, and anemia. The primary reason for weakness in this area is the fact that the normally, very adequate, *perineal sling* which supports the entire weight of the small intestines, has been removed and replaced by a new pelvic floor which is at first very weak and thin peritoneum, and later is scar tissue which is unable to support the weight of the intra-abdominal contents.

SYMPTOMS

Predominant complaints in reported and personal cases are perineal pressure, pain, and a perineal *bulge*. The onset may be at any time after surgery; more commonly occurring 12 months or longer, postoperatively. These patients have considerable pain when sitting down and a feeling of pressure when walking. The diagnosis is obvious in all cases that have been reported and is made by the patients themselves, when they complain of a perineal *bulge*. There are probably many cases that are overlooked because they are asymptomatic and mild, and most re-examinations are done either in the lithotomy or Sims positions. In order to evaluate properly the degree of perineal support present the patient must be examined in a standing position.

The following cases represent 1 personally seen, 2 abstracted from the records of Charity Hospital in New Orleans, Louisiana and 1 from the Ochsner Foundation Hospital, New Orleans, Louisiana.

CASE REPORTS

Case 1. Mrs. A. L. B., a white woman, was admitted to the Charity Hospital of Louisiana in New Orleans for the first time in July 1938. A diagnosis of adenocarcinoma of the rectum

was made at this time. In August 1938 a Devine colostomy was made, followed in one month by a Kraske posterior resection of the rectum. This procedure necessitates removal of the coccyx and lower sacrum in removing the growth. She did very well postoperatively and made an uneventful recovery.

Eight years later in January 1947 she was admitted complaining of persistent perineal drainage. There was a loss of weight of 30 pounds; there was drainage from the *blind* colon loop on the left. On Jan. 18, 1947 the colon, from the midtransverse to and including the sigmoid colon was removed. The perineum was explored and all sinuses removed. Exploration revealed a sacral hernia between the remaining sigmoid and the sacrum. The hernia, with redundant skin, was excised and the perineal wound was packed open with iodoform gauze.

The perineal hernia recurred 1 year later and 18 months later she was readmitted to the hospital complaining of a perineal bulge and pain when sitting down. In June 1949, using a perineal approach, the hernia was repaired by excising the hernial sac and ligating it as high as possible. The floor was reconstructed by using all available fibrous tissue and levator ani muscles and by reinforcing this closure with flaps of gluteus maximus muscle and fascia. The skin was closed tightly around a Penrose drain. Recovery was excellent and on examination in March 1952 there was no recurrence of the perineal hernia.

Case 2. Mr. T. M., a 58 year old white man was admitted to the Charity Hospital for the first time on Aug. 6, 1949. He gave a history of having had an abdominoperineal resection for carcinoma of the rectum approximately 20 years previously. On the night of admission he had fallen and sustained traumatic rupture of his perineal wound with evisceration of the small bowel.

He was prepared for operation with intravenous fluids and sedation. At the time of operation, the bowel was replaced and the perineal wound was closed with quilting cotton sutures in two layers.

His postoperative course was fairly smooth although he developed wound infection and drained purulent material for several days. The infection finally subsided after treatment with penicillin, streptomycin, and supportive measures. He was discharged on August 15.

The patient was followed in the clinic with routine visits and did well until Dec. 19, 1949, at which time the bowel again was found to be presenting through the perineal wound. He was admitted to the hospital for repair of the perineal hernia.

Pertinent findings on physical examination were restricted to the abdomen and peritoneum. The abdominal wall revealed a colostomy anteriorally with two openings only one of which was functional. Through the perineal scar a loop of small bowel protruded. He was prepared for operation and explored on Jan. 6, 1950.

Under general anesthesia, the distal loop of the colon of the old incomplete abdominoperineal resection was resected. The loop was a blind pouch and was clean and normal. The coccyx apparently had been excised at the original posterior resection of his rectum. Reperitonealization was done with some difficulty because of the extensive adhesions.

The patient was placed in the Sims position and the area about the herniation excised. The dead space was then packed with gauze. Postoperatively the patient did well and the perineal wound granulated nicely. Sutures were removed on the twelfth postoperative day and he was discharged on Jan. 30, 1950. He was well and asymptomatic six months later when seen in the clinic.

Case 3. Mrs. A. C., a 73 year old white woman was admitted to the Touro Infirmary in New Orleans on Dec. 10, 1951. Her history dates back to Aug. 15, 1950, at which time she had an abdominoperineal resection for carcinoma of the rectum. Following her discharge in September 1950 she had experienced an almost constant diarrhea from her colostomy which was treated with special diets, paregoric, and other medications. A diagnosis of colitis had been made by her local physician. She had also complained of a heavy feeling in the region of the perineum which was found to be due to a perineal hernia.

She was readmitted because of melena per colostomy. Using local anesthesia the strictured colostomy opening was incised and proctoscopic examination was done. Visualization beyond 2 inches was unsuccessful due to acute angulation of the colon. Barium enemas roentgenogram showed a redundant descending colon with one area of possible recurrent carcinoma.

On Jan. 5, 1952 after appropriate preparation, she was explored abdominally. There was no evidence of recurrent carcinoma but a large redundant descending colon was found and excised. The chronic, partial obstruction had caused the mucosa to be ulcerated which was the cause of the bleeding. After completing the excision of the redundant colon, it was decided to attempt intra-abdominal repair of the perineal hernia. A new floor was formed by suturing the fundus of the uterus to the presacral fascia, and by plicating the round ligaments and the broad ligaments of the uterus. Interrupted no. 0 chromic catgut sutures were used throughout the procedure. This was not the best type of repair, but her condition would not permit the necessary posterior approach.

The postoperative course was uneventful and the patient was discharged on Jan. 20, 1952. This patient was followed at frequent intervals after her hospital discharge and about four months postoperatively a recurrence of her perineal hernia was noted. The recurrence is small, asymptomatic, and has not enlarged over a period of one year. She has no perineal complaints and will not need further treatment.

Case 4. (This case abstracted with permission of the Surgical Staff of the Ochsner Clinic.) Mrs. S. H. S. had an abdominoperineal resection for carcinoma of the rectum in September 1948. During the perineal dissection there was considerable bleeding from the right middle hemorrhoidal vessels which was controlled with some difficulty. She developed a perineal fistula in the immediate postoperative period which proved to be a urinary

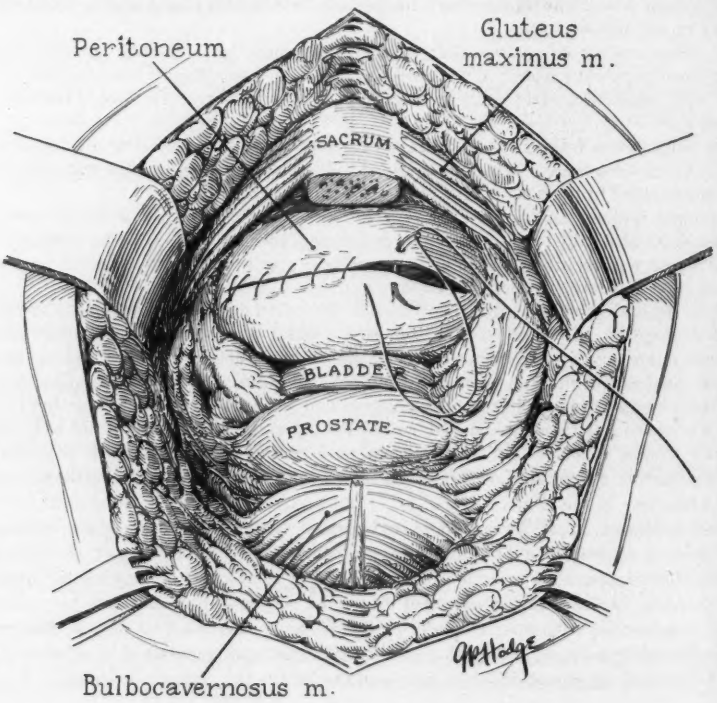

FIG. 1. High suture of hernia sac

fistula. A right nephrectomy was done about 10 days postoperatively and her course was then uneventful.

In July 1951 she was admitted to Foundation Hospital in New Orleans complaining of perineal pain which was more pronounced when she was standing, coughing, or straining. Examination revealed a cystocele and perineal hernia. The cystocele was repaired in the usual fashion. The perineal hernia was repaired from below by excision of the sac and by suturing the stumps of levator muscles over the opening.

She was readmitted in August 1952 with a recurrence of the perineal hernia. Although this hernia was not as large as the original one, perineal pain was quite severe. Careful examination revealed no recurrent carcinoma and therefore on Sept. 2, 1952 an abdominal exploration was done. The redundant peritoneum was excised and tantalum mesh was sutured around the pelvis (coccyx, pubis, and ischium). The peritoneum was sutured over this, leaving some space between the tantalum and peritoneum. It was thought that the serous accumulation would help form a fibrous tissue mass and strong pelvic floor.

The patient did well immediately after operation and was discharged approximately 14 days postoperatively. The hernia has not recurred but she still has perineal discomfort, severe enough to necessitate psychiatric care. Although the patient's response has not been good, the repair has held. The use of tantalum mesh as an adjunct should be remembered in patients in whom the fascia is not strong or strength of the repair is in doubt.

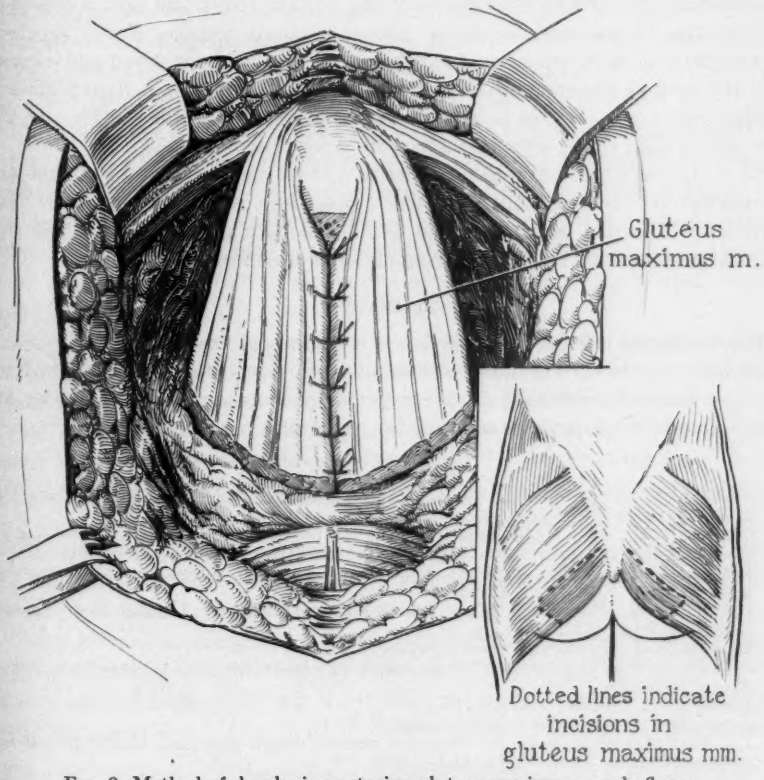

FIG. 2. Method of developing suturing gluteus maximus muscle flaps

TREATMENT

In treatment of these cases the foremost consideration is the prognosis. If abdominoperineal resection is done for palliative reasons, or if it is believed that a curative resection was not done, then to subject the patient to another procedure is certainly unnecessary. However, in those patients in whom a curative resection has been done, or in those who have survived at least three to five years with no evidence of recurrence; operative intervention should be used. Patients with perineal hernias are uncomfortable and if we consider that their life expectancy is good, repair must be attempted. One of our patients (done by Dr. Reichard Kahle) and 6 patients whose cases were reported by Gabriel[4] were repaired successfully from below by a method which we believe is the method of choice. The patients are placed in the prone position and the following technic is used:

The scar is excised and the peritoneal sac is identified and dissected out. The contents of the sac are freed from the peritoneum and reduced into the peritoneal cavity. The neck of the sac is securely closed by mattress sutures of catgut or nonabsorbable sutures such as black silk (fig. 1). The redundant skin is dissected free to identify the margins of the gluteus maximus muscle. If any portions of the levator muscles have been left behind they should be identified and sutured over the neck of the sac with interrupted black silk sutures. A flap of gluteus maximus muscle and fascia is freed and sutured together in the midline (fig. 2). The skin is then closed over this area with interrupted sutures.

All of Gabriel's patients healed primarily with no recurrence and 1 of ours was successfully repaired. Although the above procedure will certainly not be used frequently it should be remembered as an adequate procedure to repair any perineal hernia which occurs following abdominoperineal resection.

SUMMARY

The literature has been reviewed and 4 additional cases of perineal hernia have been reported. The most successful method of repair, as described by Gabriel, has been discussed. If life expectancy is good, repair should be attempted since these patients are very uncomfortable and can be helped.

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HERNIOPLASTY FOR RECURRENT INGUINAL HERNIA

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There has been a decided change in the technic employed in the repair of recurrent inguinal hernias. Within the past 15 years the use of ox fascia, autogenous fascial strips, and tantalum mesh have been rather commonplace as the procedures of choice. However, in the past five years these methods of repair have been discarded by most authorities, and in their place they are utilizing the basic anatomy to the fullest extent by adapting the surgical procedure to meet the existing anatomic conditions and type of recurrence. With a better and more thorough understanding of the surgical anatomy of the inguinal region, and with the wider use of fine nonabsorbable suture material, the repair of recurrent inguinal hernias has been made without the additional aid of autogenous fascial strips or of foreign material, such as tantalum mesh.

It is apparent too, that progress, has been made in this field during the past 10 years in lowering the recurrence rate. However, the recurrence rate on a countrywise basis remains much too high. According to a review of the literature on this subject, Stein and Casten[13] concluded that the recurrence rate varied from 25 to 35 per cent. It is difficult to assess the recurrence rates in many reported series. This is based upon our inability to determine from the reports the manner in which the rates were calculated. Frequently the follow-up period was too short to be of any statistical value. Many reports were of a select group of patients and therefore did not represent all types of recurrences in patients of all age groups. These various factors are important in presenting the true nature of the problem.

Ryan[10] recently made a thorough study of their results and reported a recurrence rate of only 1.3 per cent in 150 patients who were examined and followed by physicians over a two year period. The recurrences following common methods of repair were included in a survey made by Conkling[3] in 1951. This study revealed the rates varied from 4.3 to 38 per cent.[4, 5, 6, 7, 11, 12, 14] Burton's[1] results in his own series showed a variation of from 11 to 40 per cent in the recurrence rates using a variety of repairs. Clear,[2] in a recent review of a 10 year follow-up in their series, reported a recurrence rate of 45 per cent with Gallie's fascia strips and a 33 to 50 per cent recurrence rate with an extra-aponeurotic repair. The repair in which silk was used resulted in the lowest recurrence rate.

This report is based upon a study of 100 unselected male patients who were operated upon at this hospital during a six year period for the repair of recurrent inguinal hernias. It excludes only those patients who had repairs of their hernias more than three times before. In 10, or 10 per cent, of this group the hernias were

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bilateral, making a total of 110 hernioplasties. In none of these patients was a bilateral repair done simultaneously. The opposite side was repaired between the seventh and the thirteenth postoperative days.

These operations were done by 22 members of the surgical staff, including the house officers, using the same basic technic and employing fine nonabsorbable sutures of silk or cotton. Fascial strips or grafts were not used except in 1 patient in whom a modified Wangensteen fascia lata flap was used.

AGE INCIDENCE

The patients' ages ranged from 19 to 76 years with the largest group occurring in the fifth decade, representing 39 per cent. Fourteen per cent were 60 years of age or older (table I).

TYPES OF HERNIA

All types of inguinal hernia are included in this study. The report is based upon nonselectivity. Several of the patients were operated upon for incarceration or strangulation. In 53.6 per cent, or 59 patients, the hernias were of the indirect type; in 33.6 per cent, or 37 patients, they were direct; in 10 per cent, or 11 patients, they were both direct and indirect; in 0.9 per cent, or 1 patient, it was sliding; and in 2 patients (1.8 per cent) no hernia sac was found (table II).

TABLE I

Hernia incidence according to age

Age Group	Number and Per Cent
Under 20 years	2
20–29 years	15
30–39 years	14
40–49 years	16
50–59 years	39
60–69 years	13
70–79 years	1
Total	100

TABLE II

Types of hernia

Type	Number	Per cent
Indirect	59	53.6
Direct	37	33.6
Combined	11	10.0
Sliding	1	0.91
No sac	2	1.81
Total	110	100

TABLE III

Operations in addition to hernioplasty

Type	Number
Appendectomy	1
Femoral hernioplasty	2
Orchidectomy	1
Saphenous vein ligation	1
Total	5

Sixty-nine per cent of the hernias were on the right side. In 5 (4.5 per cent) patients, additional operations were done at the time of the hernioplasties (table III).

OPERATIVE TECHNIC

The same basic technic was employed in all patients in this group, except 1 patient in whom a fascia lata graft and flap was utilized. The basic repair in all types consisted of a thorough and complete dissection of the hernia sac. This was followed by a complete reformation and fortification of the supporting structures in Hesselbach's triangle by suturing, without tension, of the aponeurotic layers of the anterior abdominal wall to the shelving edge of Poupart's ligament. The internal inguinal ring was made very snug. A four layer repair beneath the cord in Hesselbach's triangle was made. The basic technic was similar in all respects to the technic we have employed in 4,000 primary inguinal hernioplasties.[8] The cord was partially transplanted outside of the external oblique aponeurosis, it being covered only for a distance of 1 inch inferior to the internal inguinal ring. In many instances the original repairs in this group of patients with recurrent hernias were taken down in order to substitute our basic type repair. The technic of our hernia repair has been described and illustrated elsewhere.[8, 9]

In 81.8 per cent of the operations spinal anesthesia was used; in 16.4 per cent general anesthesia; and in 2 patients (1.82 per cent) a paravertebral block was used. Nonabsorbable fine silk sutures were employed in all patients except 1.

RESULTS

A one to six year follow-up was obtained in 73 (73 per cent) patients, or a total of 78 hernioplasties, of which five were bilateral. In 82 per cent of the patients in whom a follow-up was obtained the follow-up period was over two years.

There were two recurrences, or a recurrence rate of 2.56 per cent, in the group in which a follow-up was obtained. This rate was based upon the total number of hernioplasties done in which a follow-up was obtained. The recurrences developed in both patients within two years; one within six months and the second recurrence in the fifteenth month. Both patients were over 45 years of age. One patient returned to work as a truck driver at the end of six weeks, and the other returned to work as a handy man in one week. Both patients were ambulatory

TABLE IV

Analysis of recurrences

Age	Primary Hernia Type	Recurrence After Repair in Months	Type of Recurrence	Day of Ambulation	Period of Convalescence	Type of Work
46 years	Direct	6	Direct	7	6 weeks	Truck driver
54 years	Direct	15	Direct	14	1 week	Handy man

TABLE V

Postoperative complications

Type	Number	Per cent
Pulmonary		
Bronchial asthma	1	0.9
Bronchitis, acute	3	2.7
Wound		
Hematoma	4	3.6
Hematoma, cord	1	0.9
Stitch abscess	2	1.8
Genito urinary		
Cystitis	1	0.9
Epididymitis	2	1.8
Swelling, testicle	3	2.7
Urinary retention	1	0.9
Thrombophlebitis	1	0.9
Auricular fibrillation	1	0.9
Totals	20	18.1%

after the seventh postoperative day. The recurrences which were repaired were the direct type and the succeeding recurrences were the same type (table IV).

POSTOPERATIVE COMPLICATIONS

There were no deaths in this series. There were 20 (18.1 per cent) complications in the entire group. None of the patients who were ambulatory before the third postoperative day developed complications. The most frequent complications involved the hernia wound and the genito-urinary tract (table V).

AMBULATION

Thirteen (13 per cent) patients were ambulatory within 48 hours after operation without development of systemic complications other than those occurring in the wound, such as hematoma or stitch abscess. Fifty-eight were ambulatory on the seventh postoperative day and 39 on the fourteenth day. No recurrences occurred in the small group which was ambulatory early.

Our patients were instructed to refrain from heavy lifting, straining, or athletics for a period of eight weeks postoperatively.

SUMMARY AND CONCLUSIONS

A statistical analysis of the results of 78 (71 per cent) recurrent inguinal hernioplasties performed upon 73 unselected and consecutive male patients with a follow-up of 1 to 6 years of this series of 100 cases is presented; the remaining 27 patients were lost due to change of address, death from other causes, or they failed to respond to our follow-up requests.

There were two direct recurrences, or a recurrence rate of 2.56 per cent. There were 20 (18.1 per cent) postoperative complications, of which 7 (6.3 per cent) involved the wound.

It appears from this study that the use of fine nonabsorbable sutures with proper utilization of the basic anatomy as it exists in each case, without the use of fascial strips, transplants, or tantalum mesh can result in a low recurrence rate provided well established basic principles of hernioplasty are observed.

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A SPRAINED ANKLE MAY BE A MORE SERIOUS INJURY THAN A FRACTURE

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A sprained ankle is an injury of potential gravity. Yet it is often treated with halfway measures such as injection and strapping, application of an ace bandage or soaking. These methods frequently contribute to poor results. The sprain is a spontaneously commonly reduced subluxation of the ankle joint. A moderately severe true sprain can result in the formation of a painful pannus between the talus and fibula. It is the lateral side which usually is injured. Some inversion injuries to the ankle should be energetically treated.

A sprain indicates a complete tear of a ligament. In the case of the lateral side of the ankle the sprain results in a subluxation which is spontaneously reduced before it is seen by the clinician.[1-10]

A sprained ankle can result in prolonged and sometimes permanent disability. One might ask, what is the cause of this prolonged temporary and sometimes permanent disability which often occurs following so-called sprains of the ankle? Fifty-one cases have been studied.[7] Twenty-three of these patients were shown to have complete tears of one or more components of the lateral ligament of the ankle by roentgenogram of the ankle with the joint in forced inversion and with the patient under general anesthesia. A roentgenogram was made of the opposite side in these patients for comparison, since it is known that a few degrees of instability is normal in some ankles.[2, 3, 6] All of these patients were treated with walking casts and there was no permanent disability in any of this group. On the other hand, 24 of the patients who had roentgenographically stable ankles after examination under general anesthesia were treated with novocain injection and strapping. Seven of these 24 had persistent swelling, insecurity and pain in the ankle. Four of the 51 cases studied were recurrent injuries. One of these four ankles, in a young man age 23, showed wear and tear changes as demonstrated by the roentgenogram.

Two explanations of the reason that 7 of the 24 patients who had stable ankles and still had symptoms are available. Wolin, and associates[9] have shown that, following a strain or a mild sprain, a scar of *hyalinized connective tissue* forms on occasion between the talus and the fibula. They believe that this is the locus of the origin of pain in these ankles. They have cured their patients of this condition with excision of the scar tissue. Judging from those patients treated by me, who had roentgenographically stable ankles, but residual symptoms, the formation of this *scar tissue mass* can be avoided by proper immobilization. A second explanation is that there is instability in the sagittal plane of the ankle. This phenomenon is well demonstrated in the 2 cases reported by Dr. Anderson, LeCocq and LeCocq.[1]

FIG. 1 Cadaver specimen with soft parts of the ankle other than ligaments removed. Note that with foot in equinus the anterior talo-fibular ligament runs perpendicular to the long axis of the talus, whereas the calcaneo-fibular ligament lies almost parallel to it.

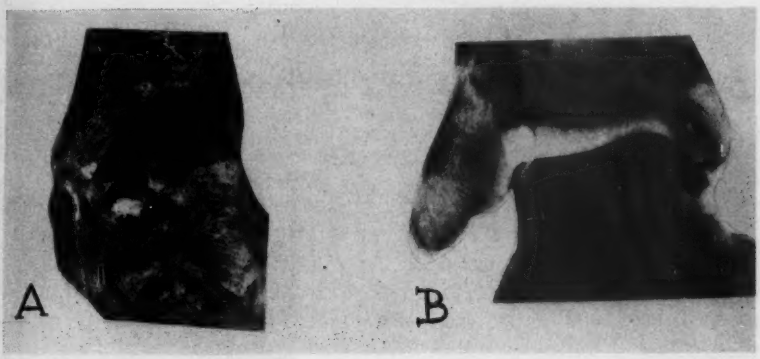

FIG. 2 A. Joint instability resulting when anterior talo-fibular ligament is cut. B. Roentgenogram of the ankle.

Let us now turn our attention to the commonest type of so-called *ankle sprain* and its mechanism. The lateral ligament of the ankle is a flimsy structure in comparison to the deltoid ligament (fig. 1). The principal stabilizing factor of the ankle as regards inversion is the tone in the muscles controlling the tendons lateral to the ankle. Inversion injuries usually occur with the foot in plantar flexion and the small anterior talo-fibular ligament in such instances takes the brunt of the force when tendon tension is absent or overcome. When this ligament is parted instability occurs. This instability increases progressively as the continuity of the other components of the lateral ligament are destroyed (figs. 2, 3, 4).

At one time it was suggested by me that every patient with a severely sprained ankle be anesthetized and an attempt made to determine whether or not the ankle was stable roentgenographically. This procedure, in my present opinion, is neither practical nor desirable. In an acute sprain of the ankle, if there is

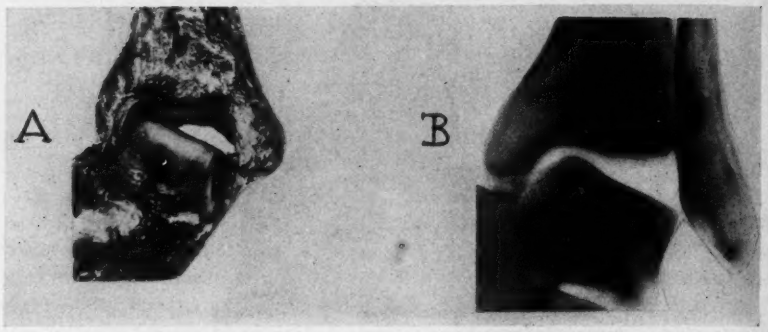

FIG. 3 A. Both anterior talo-fibular and fibular calcaneal ligaments have been cut.
B. Roentgenogram of figure 3 A.

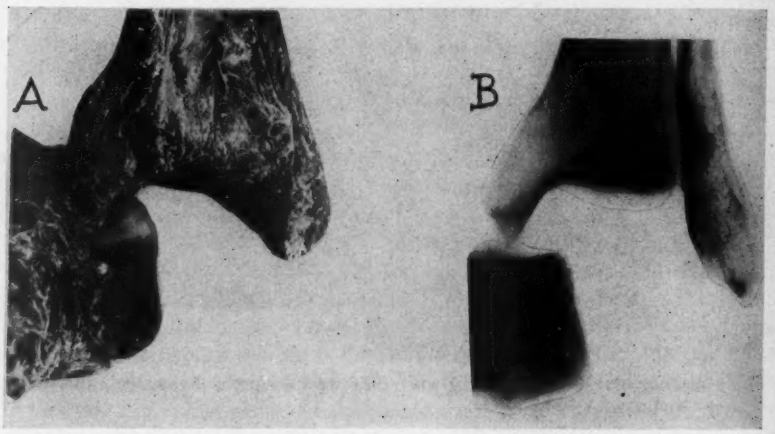

FIG. 4 A. Gross instability resulting from complete severance of soft parts on lateral aspect of ankle.
B. Roentgenogram of figure 4 A.

marked ecchymosis and swelling with tenderness over the lateral ligament of the ankle, particularly over the anterior talo-fibular ligament, I believe the patient should have a walking cast applied for a period of three weeks. If at the end of this time the ankle is no longer tender or discolored, motion should be started. If, on the other hand, there still is tenderness, cast immobilization should be continued for another three weeks. With this course of treatment it has been possible in a large series of sprained ankles routinely to obtain complete recoveries.

As regards the chronic recurring sprain of the ankle, or the *weak ankle* as it is described to you by the patient, the use of a quarter inch wedge on the lateral aspect of the heel of the shoe, if the patient does not have a tendency toward valgus of the heel, gives the patient much relief. If conservative measures fail

it is possible to reconstruct the lateral ligament of the ankle to stabilize a chronic recurring dislocating ankle.[10]

SUMMARY

The layman's aphorism a *sprain is worse than a break* indicates the importance of active treatment by plaster immobilization of some ankle sprains. These sprains actually are spontaneously reduced subluxations of the ankle. Another large group with severe injuries which cannot be shown to be unstable by roentgenographic examination, if not actively treated, eventuate in the formation of a painful scar between the fibula and talus. Hence severe sprains of the ankle should be energetically treated with weight bearing plaster immobilization. Minimal sprains can be treated by novocain injection or ethyl chloride freezing followed by strapping.

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PLASTIC IMMOBILIZATION: A NEW METHOD

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Since 1934 it has been my desire to find a water-proof material for immobilization purposes. At first, efforts were made to cover plaster of paris splints or bivalve casts with cellulose acetate solution.[1] This device was abandoned because the plaster often cracked after prolonged use, permitting wetting with softening and disintegration of the splints or casts. Any such coating is unsatisfactory also because it tends to make the cast impervious to air and fluids. Then followed the development of molded plastic supports made by pouring Lucite over a plaster mold, but this proved impractical because of the labor and expense involved. A material satisfactory in many respects then was found in a glass cloth and resin mixture,[2] but this still required the use of a bivalve cast and mold.

The first plastic material suitable for direct application in the form of a cast was developed through further research in resins. Among this class of synthetics there are the so-called melamine resins which are used industrially to bond together thin layers of such materials as plywood, asbestos and glass fiber. The products have a hard, smooth surface resistant to heat and water. These industrial uses suggested that the resin might serve to fuse together the setting particles of plaster of paris into a strong, waterproof material. For this purpose a solution of 35 per cent of the resin known as *Melmac 405* with 3 per cent by weight ammonium chloride as polymerization catalyst was prepared. Ordinary plaster of paris bandages soaked in this solution and applied directly to the patient set quickly in a cast that proved to have many advantages. It was not only waterproof but also so much stronger than ordinary plaster that casts could be applied a half to a quarter of the usual thickness. The result was a very light cast which easily could be removed with a hand cutter.

The material appeared to have a very low index of sensitivity, producing a mild skin reaction in only about 1 in every 1000 cases. The only objectionable features of this resin solution were some dripping of the concentrated melamine solution, with occasional straining, and the necessity for using disposable containers because it was thought—although as far as I know never proved—that sediment remaining in the bucket might cause some clogging of drains.

A plastic bandage which has been developed not only overcomes these inconveniences, but also meets all requirements of a plastic type of immobilization. In this bandage, crinoline is coated with a mixture of plaster of paris, Melmac resin, and a catalyst, prepared by a special method.† This bandage is ready to apply, requiring only a few seconds' wetting in water.

* Appreciation to the staff of Georgetown University for their assistance in this work.

† The plastic bandages used in this investigation were developed by and supplied through the courtesy of Davis and Geck, Inc., a unit of American Cyanamid Company, Danbury, Conn. They will be manufactured and distributed by this organization through the surgical trade under the name of Melmac resin plaster of paris bandages.

The culmination of the long search for a practical method of plastic immobilization prompts this preliminary report, based on its use in the first 126 cases. This series includes in all 143 casts applied by 12 orthopedic surgeons in several Washington hospitals.

Comparative Strength of the New Cast. Extensive laboratory investigations have demonstrated the much superior physical characteristics of the melamine resin cast in its more or less optimum formulation as used here.

The strength of a plain plaster of paris cast is always much less than that of the melamine resin plaster of paris cast; the strength of the former is largely dependent upon atmospheric conditions. Even under relatively dry atmospheric conditions, four hours after preparation the resin cast requires two to four times the stress to cause permanent deformation when compared with plain plaster casts, bandage for bandage. At this point the strength of the resin cast is 85 to 162 per cent greater weight for weight. On the other hand a completely wet and fully set resin cast requires from 4 to over 10 times the stress to cause armanent deformation as compared with a plain plaster cast, bandage for bandage. Under these conditions the strength of the resin cast is from 240 to 700 per cent greater weight for weight.

In hot humid weather even the strongest plain casts which we have tested four hours after preparation are permanently deformed at about one-third the stress required to deform a resin cast. Furthermore, under these humid conditions the plain cast does not increase in strength between 4 and 24 hours, while a resin cast under the same conditions becomes over 4½ times as strong as the strongest plain cast at 24 hours. In hot, humid weather an ordinary plaster cast retains as a maximum only 36 per cent of the strength it attains after 24 hours in a dry atmosphere, while the resin cast retains 93 per cent of its strength under these conditions. Thus, under humid conditions after 24 hours the resin cast can be at least 370 per cent stronger bandage for bandage, and 260 per cent stronger weight for weight.

Humid conditions make for a heavy, soggy, soft, weak plain cast that is uncomfortable and irritating to the patient while the properties of the resin cast are relatively independent of atmospheric humidity.

Method of Application. Because of its much superior strength, the Melmac bandage can be applied in one-half to one-quarter the thickness of plain plaster casts. The surgeon should, therefore, expect to use only one-third to one-half the usual number of rolls for each type of cast (table I). The rolls when placed in water wet immediately. One does not let the bandage stand in the bucket until it stops bubbling as is the case with plaster of paris, but holds it in the water until the bandage is soft, which requires a count of not more than five seconds. The bandage is then wrung out thoroughly. With this plastic, the rate of setting depends to a slight degree on the quantity of water left in the bandage. The drier the bandage is wrung, the more rapidly it sets. Thorough wringing has the additional advantage of neatness; there is no dripping on the patient, or spotting of floor and sheets. When wrung out, the bandage has a good creamy feel, and is very malleable.

TABLE I

Type of cast & number of bandages required

Type of Cast	No. of Casts Applied	Melmac Bandages Required	
		Usual No.	Usual Size in Width
			inches
Minerva or Calot Jackets	4	10	4
Body Casts	11	6	4
Velpeau	4	3	4
Shoulder Spica	2	7	4
Long Arm Cast	15	3	3
Short Arm Cast	11	2	3
Single Hip Spica	7	10	4
Double Hip Spica	4	11	4
Long Leg Cast	35	4	4
Short Leg Cast	7	3	3
Knee Cast	1	3	4
Boot Casts	12	2	3
Club Foot Casts	11	2	3
Splints	**No. of Splints**		
Forearm	8	1	4
Long Leg	1	1	4
Dorsal	1	1	4
Knee	1	3	3
Toe	1	½	4
Wrist	2	1	3
Hand	3	1	3
Total	141	Reactions—None	

It might be mentioned that with this new bandage there is no appreciable loss of plaster or plastic, either in the wrapper or in wetting. What little residue there may be in the bucket after soaking may be poured into any drain with no danger whatsoever of clogging, and the bucket needs only to be rinsed with water.

In applying the new melamine plaster bandage, care must be taken not to make the cast too thick. This cannot be overemphasized, since many of its advantages depend upon proper application. The cast should be light and thin. Where added strength is needed, as about the joints, one or two added figure-of-eight wrappings will be sufficient. The bandage sets so hard that, when well rubbed, it has a porcelain-like consistency.

Observations on Use of the Cast. This cast has been found suitable for all types of immobilization. The material hardens quickly, with such high early strength that patients can often be removed from the table in a short time. A patient with a double hip spica, for example, was removed in 15 minutes, and a body cast case within 30 minutes. Water does not soften or in any way damage this cast. The patient can wash or even shower freely. Whirlpool baths and other pre-

scribed hydrotherapy can be started early. The patient can wear a skin-tight cast, soak the extremities in water and exercise the parts, distal and proximal. This cast will not soften by absorption of wound exudates or urine. In spite of its excellent strength and waterproof properties the resin cast is amply pervious to air, perspiration and wound exudate so that it is comfortable in hot weather and does not cause maceration.

The lightness of the cast not only makes it easier for hospital personnel to handle the patient, but reduces immobility, aiding the patient in more rapid return to normal function of the parts. Ambulant patients do not find this cast so handicapping to their usual activities as plaster of paris, because of its strength and lightness.

Since the cast is thin, roentgenograms taken for check-ups are clearer, and can be read more accurately. If necessary, a window can be cut in the cast for an operation or dressing, and the cast can be repaired easily.

Reactions. To date, no reaction of any kind has been observed with the new bandage. Of particular interest is the fact that 3 patients who showed some skin irritation with the older resin-dipped bandages have been patch tested with the new material, and showed no reaction.* This suggests that the mild irritation seen occasionally when the solution was used may have been due to the high concentration of resin then required, from which it was difficult to remove formaldehyde. Since a concentrated solution is no longer used, the possibility of any type of skin reaction, in either operator or patient, should be practically eliminated.

Method of Removal. The cast can be removed very easily and quickly, with either scissors or a Stille type cast cutter. The material has a tendency to brittleness, which is an advantage in removal. In cutting, one twists the cutter slightly sideways, and the cast cracks ahead of the cutter. Most casts removed so far have required only three to five minutes. Body casts have been removed in five to seven minutes with a hand cutter. Electric cast cutters designed for use on ordinary plaster casts are at a disadvantage in removing the resin cast because of its hardness and tendency to heat up and become dull. Some improvement in this type of instrument may be indicated in order to improve its effectiveness. The hand cutter is much easier to use on this cast than on any other.

SUMMARY

A plastic prepared bandage has been developed that forms a waterproof, light, strong cast that will not soften by absorption of drainage from beneath or moisture from without. Additional advantages are ease of wetting, and quick setting without danger of staining sheets or clogging drains.

The chance of skin irritation, which was already at a minimum, should now be negligible.

* At the present time, 4 patients who have shown some sensitivity to the resin-dipped bandage have been wearing casts of the new material for 10 weeks. No reaction has developed in any of these patients.

The cast is always several times as strong as unfortified plaster of paris and its strength—in contrast to that of plain casts—is relatively independent of atmospheric conditions.

Splints or shells can be prepared easily and quickly for paralytic patients, and skin-tight casts can be used for hydrotherapy and for early rehabilitation of fracture cases and postoperative patients.

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EDITORIAL

THE SAFETY OF PLASMA

Pooled plasma which has little or no risk of transmitting homologous serum jaundice can be prepared, providing it is stored in a liquid state at room temperature for six months or longer before use. No virus will survive prolonged periods of room temperature storage in a liquid cell-free medium and data are now available which demonstrate that this principle applies also to the virus of homologous serum jaundice.

This was first pointed out by the University of Chicago group where this procedure was suggested as a means for preparing nonicterogenic plasma. This group experienced no cases of homologous serum jaundice attributable to the plasma given to 1,360 patients. At the same time, they observed 44 cases of homologous serum jaundice from whole blood given to 11,660 patients. All patients concerned in this study were observed for a minimum of six months following the last transfusion.

Recently a survey conducted by the American Association of Blood Banks coupled with data from additional sources, disclosed the following experience from 217,134 transfusions of pooled plasma. Twenty-five thousand two hundred and forty-seven units of this plasma, or 13 per cent of the total plasma given, had been stored in the lyophilized, frozen or refrigerated state. Two hundred and fifty-nine of the patients receiving these types of plasma were known to have developed homologous serum jaundice. The remaining 87 per cent or 191,887 units of plasma had been stored at room temperature in a liquid state for three to nine months prior to administration to patients. From this much larger group of transfusions there were no patients known to have developed homologous serum jaundice.

If, however, plasma is drawn from donors who are sick at the time with homologous serum jaundice—Murray has demonstrated—a plasma with much greater infectivity is obtained. Murray finds that the attack rate for this particular plasma is reduced from 52 per cent to 5 per cent after six month's storage at room temperature in a liquid state. This observation confirms the fact that the virus of this highly infective plasma responds to effects of room temperature storage in a manner similar to that of ordinary plasma which is contaminated by the less infective virus. It is clear, however, that to accomplish complete inactivation of the highly infective virus of Murray, a storage period slightly longer than six months may be necessary. Whether a short extension of the room temperature storage period beyond six months should be applied to all plasma to insure safety depends upon whether the present donor selection policies are to be continued.

There are no important physicochemical changes that occur during the first two and one-half years of room temperature storage of liquid plasma that effect its usefulness in transfusion for the treatment or prevention of shock or malnutrition. Lozner in 1944 found little alteration in the proteins of plasma

stored for a three year period. More recently, Barron of the University of Chicago, and Chanutin of the University of Virginia, examined citrated pooled plasma which had been stored at room temperature for intervals extending to 10½ years and have come to similar conclusions. According to Chanutin, "Aged plasma collected in citrate (without acid-citrate-dextrose solution) showed comparatively small changes in the electrophoretic pattern." Some change does occur between the third and fifth year of room temperature storage and affects primarily the globulin fractions. A small percentage of the globulins split into smaller molecules of the molecular range of albumin. This cleavage Lozner found to result in a plasma of greater oncotic pressure than it originally possessed. Between the fifth and tenth years of storage this product underwent little additional change.

The clotting factors are rapidly lost from pooled plasma stored at room temperature. Some of these also deteriorate in frozen or lyophilized plasma, but at a rate much slower than in liquid plasma. This is not objectionable since fresh plasma is of little benefit in the treatment of abnormal bleeding disorders other than hemophilia. The risk of homologous serum jaundice from freshly prepared plasma when used in the treatment of hemophilia is reduced by not pooling such a product. The coagulation disorders that do respond to the clotting components of plasma are best treated by the administration of the appropriate plasma fractions.

The virus dies out long before any important change occurs in plasma proteins. Therefore if desired liquid pooled plasma can be safely dried between the sixth and thirtieth month of room temperature storage without fear that severe alterations in the plasma proteins have occurred. For the community hospital blood bank where large stock piles of plasma are not required, plasma can be maintained at room temperature without resorting to lyophilization.

Drying, freezing or refrigerating of pooled plasma preserves the virus of homologous serum jaundice when present. These procedures should not be employed in the preparation of plasma for clinical use unless it has been rendered free of virus before such storage procedures are instituted. Indeed, these have been the procedures most often used in the preservation of viruses. Since most pooled plasma has been preserved by one of these methods, it is understandable why such plasma continues to produce homologous serum jaundice.

Exposure of liquid plasma to ultra violet light, while probably reducing the attack rate and extending the length of the incubation period, has not been completely successful in eradicating the hazards of homologous serum jaundice. On the other hand ultra violet light exposure of plasma has proved worth while as a means of killing contaminating bacteria. The several virucidal chemical agents that have been added to plasma, have either failed to destroy the virus of homologous serum jaundice or have in themselves proved too toxic for general use.

Blood and plasma play a vital role in the rehabilitation of the patient who is unable to eat. These two products provide a most efficient means for the prevention or treatment of hypoproteinemia in this type of malnourished patient.

Their nutritive value is established by both experimental and clinical data. Terry and Whipple were able to maintain dogs in a good nutritive state for at least three months when intravenous plasma was the only source of nitrogen. Reports from other laboratories arrive at similar conclusions for both the experimental animal and man. Yuile and associates believe that plasma administered intravenously can be incorporated into organ protein without its breaking down to the amino acid level.

Hypoproteinemia is most economically treated in patients who are unable to eat by the administration of blood until the circulating hemoglobin and blood volume are restored to normal. Thereafter, the administration of adequate amounts of plasma will correct any persisting hypoproteinemia. By such procedures it is possible to prepare malnourished patients for operative procedures and to reduce materially surgical-risk in debilitated patients.

The usefulness of the fractionation products of plasma for certain specific disease states is well established. There are, however, certain limitations of the fractionation procedure that are not generally appreciated. The primary limitation is the poor yield of transfusable protein. More than half of the proteins available in plasma are either lost in the current fractionation process or are no longer suitable for transfusion purposes. Since plasma is as useful as serum albumin in the treatment of most kinds of shock and hypoproteinemia, an important economy of our transfusable protein resources can be achieved by the use of whole plasma wherever possible.

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BOOK REVIEWS

The editors of The American Surgeon will at all times welcome new books in the field of surgery and will acknowledge their receipt in these pages. The editors do not, however, agree to review all books that have been submitted without solicitation.

Peripheral Nerve Injuries. By WEBB HAYMAKER, M.D., Chief, Neuropathology Section Armed Forces Institute of Pathology and BARNES WOODHALL, M.D., Professor of Neurosurgery, Duke University School of Medicine. 2nd edition, 333 pages, 272 illustrations. Philadelphia, W. B. Saunders Co., 1953.

The second edition of this monograph continues the high level of excellence previously established. The four sections deal with principles of innervation, examination of the patient, classification of nerve injuries and finally, clinical features of the various nerve injuries. The text is lucidly written and clearly illustrated. It is complete without being overly long.

For the surgeon dealing with peripheral nerve injuries, civilian or military, this book is highly recommended.

CHARLES E. BRACKETT, JR., M.D.

Surgical Pathology. By PETER A. HERBUT, M.D., Professor of Pathology, Jefferson Medica, College, and Director of Clinical Laboratories, Jefferson Medical College Hospitall Philadelphia, Pennsylvania. Second Edition, Thoroughly Revised, with 528 Illustrations Philadelphia, Lea and Febiger, 1954; Price $14.00.

This volume contains 864 pages of textual and bibliographic material. In his preface to the first edition the author indicates that his book is designed to serve as a study source primarily for those men preparing for examination by one of the specialty certifying boards. In the arrangement of material he has kept two ideas in mind, "(1) to treat the subject from a regional viewpoint; and (2) to summarize, as far as possible, the current ideas from the current literature".

Diseases are considered under the headings; Congenital Anomalies, Inflammations, Tumors, and Mechanical Disturbances. This makes for a logical and concise presentation. It is coupled with a preliminary discussion of embryology and anatomy at the beginning of each chapter. An adequate bibliography appears at the end of each chapter.

The illustrations are numerous in this book and of good quality, although the photomicrographs far surpass the gross specimen photographs in quality.

New chapters on the Adrenals and the Central Nervous System have been added to this second edition, and several chapters have been extended to parallel extensions of the applications of surgery.

In general this is believed to be a very valuable book, and accomplishes well the aim of its author.

THOMAS G. ORR, JR., M.D.

Peptic Ulcer. by C. F. W. ILLINGWORTH, C.B.E., M.D., Ch.M., F.R.C.S.E., F.R.F.P.S. (Glas.), Regius Professor of Surgery, University of Glasgow, Surgeon, Western Infirmary, Glasgow. Edinburgh and London, E. & S. Livingstone, Ltd., 1953, Baltimore, The Williams & Wilkins Co. Price $8.50.

This excellent monograph on peptic ulcer will be especially welcomed by all those interested in this disease who are familiar with the previous writings on this subject by this British author. Although the author is a surgeon, the subject has been covered in all phases and only 23 of the 285 pages are devoted to surgical treatment. There are excellent chapters on gastric physiology and pathology as well as the experimental aspects of the subject.

The chapters dealing with etiology and incidence of peptic ulcer are especially well presented with a discussion of how statistical studies of the changing factors of sex, race, family, age, economics, occupation and geography may ultimately lead to a sound understanding of cause and prevention of the disease.

Each chapter is followed by a small but well selected list of references and the author states that he has quoted with discrimination, preferring a few reliable facts to a host of opinions.

The material is well organized and illustrated and the quality of the writing is exemplified by the following vivid description of ulcer pain: "Few patients describe the pain as severe, and it rarely approaches the agony of, say, a bad toothache or a crushed finger. It is nothing like the rasping pain of pleurisy, the anguish of a colic or the sword thrust of a perforation. It harrows and afflicts rather by its persistence and iteration than its harshness. It is a dreary dismal dark grey kind of pain, most faithfully limned in the woebegone expression of its victims."

This volume will be a valuable addition to the library of every physician treating patients with peptic ulcer.

GEORGE A. HIGGINS, M.D.

Experimental Surgery Including Surgical Physiology. By J. MARKOWITZ, M.B.E., M.B. (Tor.), Ph.D., M.S. in Exp. Surg. (Minn.), Associate Professor of Physiology, University of Toronto, Visiting Professor of Physiology, Ontario Veterinary College, Guelph, Ont. In Collaboration with: J. Archibald, D.V.M., M.V.Sc., Professor and Head of the Division of Small Animal Medicine and Surgery, Ontario Veterinary College, Guelph, Ont. and H. G. Downie, D.V.M., M.S. (Cornell), M.V.Sc., Assistant Professor of Physiology, Ontario Veterinary College, Guelph, Ont., Third Edition, Baltimore, Maryland, The Williams & Wilkins Company, 1954. Price $10.00.

In the introductory remarks the author states that "to be experimentally minded is exceedingly valuable to the clinician since, better than anything else, it teaches him to weigh clinical evidence". This statement emphasizes the valuable material contained in this book.

Anesthesia and equipment for use in anesthesia of animals, and the armamentarium for animal experimentation are discussed in detail.

The beginner in surgery is taught the standard methods of suturing wounds and tying knots. There is a chapter on surgical exercises suitable for students, interns, and residents who aspire to be initiated in experimental work in surgery, and for those who are training their hands for clinical operative surgery.

Many technics of standard procedures in the surgery of the alimentary tract are described and illustrated. Methods used in making physiologic experiments are presented. The Heidenhain pouch, Dragstedt's methods of studying the gastric secretions, the Pavlov pouch, methods of studying the surgical physiology of intestinal obstruction, diseases of the esophagus, pancreas, biliary tract, liver, and circulatory system, peritonitis, and transplantation of organs are fully described. The mechanical heart and mechanical kidney are included.

This is not only a book of surgical technic, but also an outline of surgical physiology. It is a useful guide for the beginner in operative surgery and for anyone interested in experimental surgery.

To make the text complete from the standpoint of experimental surgery there is a chapter on the care and feeding of animals.

THOMAS G. ORR, M.D.

The Thyroid. A Physiological, Pathological, Clinical, and Surgical Study. By T. LEVITT, M.A., F.R.C.S. (Eng.), F.R.C.S (Ed.), F.R.C.S.I., Hunterian Professor of the Royal College of Surgeons of England; Lecturer in Surgery to the Fellowship of Post-graduate Medicine at the New End Hospital, Hampstead, London; sometime Surgeon, Paddington

Hospital, London; First Surgical Assistant, Thyroid Clinic, New End Hospital, London; Demonstrator in Anatomy, Middlesex Hospital, University of London., E. & S. Livingstone, Ltd., Edinburgh and London, Baltimore, Maryland, The Williams & Wilkins Co., 1954. Price $20.00.

This encyclopedic treatise upon the thyroid is outstanding in its thoroughly complete treatment of the subject.

Twenty-eight chapters are grouped in sections: The Natural History of Thyroid Disease; Clinical Aspects of Thyroid Disease; and Treatment. This points up the scope of coverage in the text.

The critical reader may find many areas of controversy but he cannot fail to be impressed with the tremendous amount of work and preparation that has been required to produce this book. The clinicopathologic study of 2,114 thyroidectomies by the author should form sufficient background for impressive and convincing discussion, and this has been supplemented by excellent presentations of normal and abnormal physiology, unique treatment of original descriptive material upon Hashimoto's and Riedel's diseases and one of the more lucid offerings on pretibial myxedema. The use of charts, diagrams, and photographs, of both black and white as well as colored varieties, has added much to the richness of this volume. Finally, the author has kept thoroughly abreast in the treatment of thyroid disease with a section on modern surgical technic and the use of radio-iodine.

Surely no medical library is complete without this splendid book.

MAHLON H. DELP, M.D.

BOOKS RECEIVED

Books received are acknowledged in this section, and such acknowledgement must be regarded as a sufficient return for the courtesy of the sender. Selections will be made for review in the interests of our readers and as space permits.

1954 Medical Progress. A Review of Medical Advances During 1953. By MORRIS FISHBEIN, M.D., Editor, New York, Toronto, The Blakiston Company, Inc. Price $5.00.

Carcinoma of the Colon. By LELAND S. MCKITTRICK, B.S., M.D., F.A.C.S., Clinical Professor of Surgery, Harvard Medical School, Surgeon-in-Chief, New England Deaconess Hospital; Consulting Visiting Surgeon, Massachusetts General Hospital; Consultant in Surgery, Peter Bent Brigham Hospital and FRANK C. WHEELOCK, Jr., A.B., M.D., F.A.C.S., Assistant in Surgery, Massachusetts General Hospital; Assistant in Surgery; Harvard Medical School. Springfield, Illinois, U.S.A., Charles C. Thomas, Publisher; Price $3.25.